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ARCHIVES OF OTOLARYNGOLOGY

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VOCAL AND VERBAL SYNDROMES

THEIR RHINOLARYNGOLOGIC SIGNIFICANCE

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I have often wondered how many laryngologists realize the value of alterations in the voice as a preliminary diagnostic aid in conditions of the throat—how many, in making a diagnosis, use their auditory as well as their visual sense. I believe that the laryngologist—if he possesses a trained ear—should be well on the road to a diagnosis as soon as he hears his patient speak, because I have found that just as there are characteristic physical alterations peculiar to certain pathologic conditions of the larynx, there are also characteristic *vocal* alterations.

Before developing this thesis, I wish to recall some of the basic facts of voice and speech production. Voice, as distinguished from speech, is simply the production of sound through the medium of expired air. A normal voice has certain definite characteristics: a pitch appropriate to age and sex, adequate volume, tonal clarity, resonance, rhythm and a direct attack.

Speech is articulated voice and may be described as a systematic grouping of purposive organized sounds conveying complex meaning. The normal sounds of speech require not only the vocal cords and pulmonary apparatus but the oral and nasal cavities and the lips, tongue and teeth. The waves set up by the vibrations of the vocal cords are modified by these structures and thus differentiated into the various sounds of speech. Further, these structures are under the control of the central nervous system, and ideas arising there are given expression in speech through association fibers, the speech centers and the centers controlling the peripheral muscles involved. The sense of hearing also is highly important for the effective performance of speech, and the normal and uninterrupted flow of speech depends on the proper functioning and coordination of all of these structures, central and peripheral.

This paper and those that follow were read as part of a Symposium on Vocal Defects at the Forty-Fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, May 11, 1939.

Disturbances of the speech and language functions constitute much too complicated a field to be discussed here, however, although they, too, have their laryngologic significance. Here only those vocal alterations will be considered that arise from laryngeal disease.

The larynx, rather incorrectly, is generally spoken of as the organ of voice. In reality, however, it is part of the pulmonary system, and its respiratory function is primary. The abductor muscles open the glottis and allow the necessary space for breathing, while the adductor muscles bring the cords together for the secondary function of phonation.

Phonation, as the term is commonly used, means the setting in motion by the vocal cords of a column of expired air. For normal phonation the cords must approximate, draw tense and vibrate. Sounds of the pitch desired and with a certain amount of overtones are thus produced by the vocal cords. As I said, these sounds are amplified and selected in the pharynx, nose and mouth.

A minor pathologic change can cause faulty functioning of the vocal cords and interfere markedly with voice production. The most common vocal alteration arising from faulty approximation of the cords is hoarseness (trachyphonia). The common symptom of hoarseness is so generally encountered by the laryngologist that too often he perfunctorily dismisses it, failing to appreciate that it may be as important a diagnostic aid as his laryngoscopic observations.

However, the fact that vocal alterations, such as hoarseness or even aphonia, are symptoms of laryngeal disease does not mean that either larynx or cords are necessary for voice production. A much simpler organ would have been sufficient for man's vocal needs. The cat, for example, has no vocal cords but every one knows that it has a voice which—much to the nocturnal discomfort of its hearers—demonstrates a wide range of discordance.

Man also can produce a fairly normal voice without vocal cords, as the postoperative voice in the following case of keratosis demonstrates:

Case 1.—D. S., a man aged 43, complained of hoarseness of over two years' duration. Direct laryngoscopic examination by Dr. David Jones, of the Manhattan Eye, Ear and Throat Hospital, showed an irregular mass involving both cords. After biopsy of a number of specimens the condition was diagnosed as hyperkeratotic papilloma. The left cord and subsequently the right one were removed, so that now the patient has no cords whatsoever, with the exception of some fibrous tsisue on the left side.

(A recording of the patient's voice and speech was played.)

This patient's voice is rough, gruff and coarse—a muffled voice lacking in intensity and overtones. However, it is evident that the patient has no difficulty in speaking and finds his voice adequate in spite of the extensive destruction of tissue and the impairment of function. In fact, the larynx is more efficient mechanically than in other conditions in which destruction of tissue is often not so extensive.

LARYNGEAL STENOSIS

In chronic laryngeal stenosis voice and speech are often severely affected, even after dilation of the larynx has been successful. In such cases the acquisition of adequate voice and speech may be greatly enhanced by the introduction of speech and voice training along with the dilatory treatments. The following case illustrates this twofold therapy:

CASE 2.—Marie S., aged 11 years, had emergency tracheotomy for diphtheria when 20 months old. About a year previous to the time of writing, at the age of 10 years, she was brought to the bronchoscopic clinic of the Manhattan Eye, Ear and Throat Hospital for decannulation. Laryngoscopic examination at that time showed the larynx completely closed. She has been under a therapeutic regimen consisting of dilation with sounds and the use of core molds ever since.

(A recording of the patient's voice and speech made six months after dilatory treatments were instituted was played.)

At the time of the recording her voice was hardly recognizable as a human voice at all. It was a distinctly unpleasant, scraping, quacking, tracheal voice, with a pronounced inspirational stridor. In short, she sounded exactly like Donald Duck.

Speech training was instituted in conjunction with the dilatory therapy at that time. During the next three months she attended the clinic of the National Hospital for Speech Disorders twice weekly, receiving one hour's speech therapy at each visit. At the end of that period a second recording was made. The improvement noted at that time was remarkable.

(A recording of the patient's voice and speech made after three months' treatment was played.)

Her voice may now be described as adequate, although still far from normal. Tonal clarity, resonance and rhythm are now present, and the inspirational stridor is gone. Her speech is now readily understandable, but the volume is not strong because decannulation has not yet been possible. Self consciousness causes her attack to be weaker than need be, but progress thus far convinces my colleagues and me that continued therapy will produce a voice and manner of speech normal in all respects.

CANCER

When considering some of the other laryngeal involvements, one finds that in the presence of malignant conditions the laryngologist's auditory sense is especially important, for hoarseness often leads to early recognition and diagnosis of the condition.

In cases of cancer of the larynx, I have noticed a striking similarity of vocal abnormalities, whether the lesion is intrinsic or extrinsic. The next 2 cases illustrate this:

INTRINSIC CANCER OF THE LARYNX

CASE 3.—F. E., aged 54, first lost his voice over a year previous to the time of writing. At that time Dr. H. B. Orton recommended that a biopsy be done, but the patient refused, and for eleven months received no therapy whatsoever. He then came to the clinic of the National Hospital for Speech Disorders, complaining of hoarseness.

Examination disclosed an ulceration on the left arytenoid and a thickening of the left ventricle. The patient was referred back to Dr. Orton, who confirmed the tentative diagnosis of cancer. Biopsy of a specimen from the left ventricular band disclosed an intrinsic cancer of the larynx of grade III malignancy. Dr. Orton found the lesion so extensive "that to remove the whole thing by operative procedure was out of the question."

(A recording of the patient's voice and speech was played.)

EXTRINSIC CANCER OF THE LARYNX

CASE 4.—M. C., a man aged 65, had a history of hoarseness going back to 1911, at which time he had had some form of operation. His hoarseness had continued "off and on" ever since that time. Dr. Jones stated that a recent roentgen examination had disclosed a tumor of the left side of the mediastinum. Laryngoscopic examination showed marked involvement of the right side of the larynx. Biopsy revealed epidermoid carcinoma, type I. The involvement affected the right vocal chord, the left side of the mediastinum and the apex of the left lung. The condition was considered inoperable, and the patient was sent to a home.

The voices of such patients, although they often differ in other tonal qualities, tend to be characterized by wheezy undertones. In this respect the 2 cases afford striking examples. Analyzing the voices, one finds that they are both toneless, rasping and decidedly tracheal. They are muffled and strained almost to the point of being aphonic and have a peculiar quality which can be readily discerned by the trained ear. Of course, the characteristics are less well defined in the presence of the extrinsic growth but both voices have the same *wheezy undertone* and are decidedly different from the voice associated with benign conditions of the larynx.

HEMANGIOMA

The vocal changes associated with a benign condition are decidedly different from those associated with cancer. The following case of hemangioma of the right vocal cord illustrates this:

CASE 5.—H. T., a man aged 26, gave a history of hoarseness of five years' duration.

Examination of the larynx revealed a hemangioma about the size of a small pea on the right vocal cord in the midportion, involving the approximating surfaces. The hemangioma was removed, and the patient's voice is now perfectly clear.

(A recording of the patient's voice and speech before the removal of the hemangioma was played.)

The voice associated with a benign tumor does not have the wheezy quality that characterizes that in cases of a malignant growth. Although this voice is flat and raucous, with a coarse, heavy vibrato and little intonation, it does have intensity, which the voices in the cases of malignant growth lack.

TUBERCULOSIS

Inflammatory conditions of the larynx, as every one knows, are the most frequent cause of hoarseness. Acute and chronic laryngitis are the commonest of these conditions. Tuberculosis of the larynx is the next in order of prevalence. The most frequent site of the tuberculous lesion in the larynx is the posterior commissure, and in the following case this typical involvement was present.

CASE 6.—W. M., a man aged 26, when first seen at Mt. Sinai Hospital, complained of hoarseness of five weeks' duration. He had been having similar attacks of hoarseness for the past five winters.

Examination revealed a flat, slightly irregular mass involving the mesial part of the left arytenoid and the left half of the interarytenoid space. The mass crossed and ascended anteriorly into the posterior part of the right vocal cord. A biopsy revealed a tuberculous condition. A thoracic examination showed that the patient had bilateral pulmonary tuberculosis.

(A recording of the patient's voice and speech was played.)

An analysis of this voice reveals definite characteristics common to such patients. The voice is rough and has a *breathy huskiness*. Resonance and inflection remain practically normal, and volume is not affected to the degree that the rough whisper would lead one to believe. The whisper seems to be a self-protective measure which such patients unconsciously adopt in order to avoid straining their vocal mechanism. Usually they can speak loudly when it is necessary, so that the apparent weakness in most cases is disproportionate to the size of the presenting lesion.

HYSTERICAL APHONIA

For purposes of contrast, I shall now consider a case in which the vocal condition was psychogenic. Although the loss of voice originally was due to an infection, it persisted long after the local condition had completely cleared up.

CASE 7.—J. A., a woman aged 20, a student nurse, was ill for five weeks with an infection caused by a hemolytic streptococcus. She was unable to speak above a whisper. After the infection had cleared up, she continued to speak in this whispered voice, in spite of all further local therapy, for ten weeks.

When she was referred to the clinic of the National Hospital for Speech Disorders examination disclosed nothing more than a slight bowing of the vocal cords, which could in nowise account for her almost complete aphonia. It was therefore concluded that her hypophonia must rest on a functional or psychogenic basis.

(A recording of the patient's voice and speech before treatment was played.)

The patient's speech was a scarcely audible whisper. However, in such patients, the whispered voice, whether of the low or the "stage whisper" type, has no wheezy quality, nor does it sound strained, as in the inflammatory conditions. The patients seem to appreciate unconsciously that their vocal condition is an escape mechanism, and therefore they usually accept it rather complacently.

In this case the patient was readily led to understand the true meaning of her symptom, and a brief psychotherapeutic interview brought about immediate and permanent results. After one hour's treatment her voice was restored.

(A second recording of the patient's voice and speech, made after one hour's treatment, showed a marked contrast with the almost complete aphonia of the first recording.)

Her voice, although decidedly audible, was still somewhat hoarse and lacking in volume. Because of long disuse of the larynx and of anxiety regarding her vocal powers, she still lacked confidence. Three days later the patient revisited the clinic, and another recording was made.

(A third recording of the patient's voice and speech was played.)

Her voice is now normal in all respects. Through continued use of the voice, volume has been restored, and because she has gained confidence and lost her anxiety regarding her vocal abilities, the attack is no longer weak and hesitant. The patient has continued to speak normally ever since.

In conclusion, I wish to reiterate that the laryngologist should be able to differentiate between the various types of hoarseness. He should readily recognize in the voice of his patient the wheezy undertone that accompanies malignant conditions of the larynx, the breathy huskiness of tuberculosis or the characteristic vocal changes that accompany other conditions which continually come under the laryngologist's observation. In other words, if he possesses a trained ear he should, in many cases, be well on the road to a diagnosis as soon as he hears his patient speak.

61 to 63 Irving Place.

DEFECTS IN SPEECH IN RELATION TO DEFECTS IN HEARING

IRVING WILSON VOORHEES, M.D.
NEW YORK

The influence of defects in hearing and in speech on the social and economic success of civilized man is so obvious that it seems not to require any extended comment. And yet this very fact may give the reason that no thoroughly scientific study of the interrelationship of speech and hearing has been undertaken until recently.

A deaf child is put into a special school, where he is taught to convey his thoughts manually. In earlier days, it was taken for granted that one could not educate him in the broader sense, and so his training was largely vocational, and the one vocation for which every such child seemed foreordained was printing. Every deaf-mute had to become a printer, whether he wanted to or not.

Moreover, the "deaf and dumb" were naturally supposed to be "stupid," and so they were kept in a state of more or less blissful ignorance all their lives, having few continued contacts with perfectly normal men and women. No wonder they were thought to be "queer" and "abnormal" and entirely outside the bounds of the consideration and courtesy commonly accorded to "normal" men and women. The die was cast; their lot was predetermined, and, while kind and sympathetic neighbors not infrequently declared that it was "too bad" and "very unfortunate" and a "heart sorrow" to parents and relatives, it was the will of Divine Providence, and nobody could do much about it save to "put the child into an Institution."

No wonder, again, that when it became known what Anne Macy had done for the blind and deaf Helen Keller the world gasped with astonishment. Essays, articles and books came tumbling from the press, most of them stressing the "genius" of the unfortunate little girl who was so "bright" that she had actually learned to talk and hear through the sense of touch. Prof. Alexander Graham Bell was interested in the Keller child as a "phenomenon," but it is doubtful if he ever dreamed that she could be educated even in the simplest sense, to say nothing of earning a degree from Radcliffe College! It required the far seeing, painstaking, scientifically minded, ingenious Miss Macy

Read as part of a Symposium on Vocal Defects at the Forty-Fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, May 11, 1939.

to work the miracle of "restoration," and miracle it was but in a strictly scientific sense only. All the elements were present with which this great teacher might work, but they were present also in other children. Miss Macy could have wrought her miracle in some other child had this other child been lucky enough to fall under her care. It happened that Helen Keller had the "I. Q." necessary for adequate tutelage, but she was no genius in her own right. She became a genius solely and truly because of her teacher. This she has always gratefully acknowledged on every possible occasion.

Speech defects are even more common than defects in hearing. They are present in every one and become manifest under undue excitement or nervous strain. The old experiment of asking some one to repeat a difficult sentence rapidly without error always brings forth a laugh. It takes no end of repetition to state correctly that "Peter Piper picked a peck of pickled peppers" or to utter the famous line about "Sister Susie's sewing shirts for soldiers" of the turbulent World War days. Every one soon had Susie showing sirts for shoulders. It was unavoidable. The mind is capable of working with lightning-like rapidity, but the motor pathways which coordinate the muscular movements of the tongue are blessed with no such agility. Moreover, one stutters not only with one's tongue but with one's hands and feet. An awkward person is one who "gets in his own way," who stumbles, knocks over furniture or upsets his glass of water regularly at table. Who has not stuttered on the typewriter? One sets down what one thinks is a pretty sentence, but when one comes to revise it letters are turned around out of their natural order in a word, words are omitted entirely or a terminal letter or syllable is missing. It is compromising to one's sense of intellectual superiority. Yes, one stutters not only with one's tongue but with one's body. For some, perfect coordination can never be acquired. One can, of course, improve one's status, but one can never perfect it.

Although defects of speech and hearing are closely allied at times, they are often separate and independent. Errors in speech are, indeed, so common that they outnumber defects in hearing two to one or perhaps in an even greater ratio. There is the example of the lapsus linguae of a learned college professor of literature who wished to excoriate the "blood and thunder" type of novel but who inadvertently called it the "thud and blunder" school. The story goes that he was, indeed, proud of his bon mot and more than willing to let it stand unchanged. This is, to be sure, not a bona fide example of a speech defect per se, but it illustrates the tongue twister to a nicety.

But the profoundly deafened person always has trouble with voice production, because he does not know whether he is speaking too loudly or too softly, distinctly or indistinctly. He probably has more trouble

with pitch than with quality. In aural testing there is a "reading test" which was developed by Bárány while I was in Vienna many years ago. For this, one needs a noise apparatus or two of them if testing the voice of a normally hearing subject for conversational effect. The subject is asked to read and to continue reading without a stop. As soon as one has heard enough of the ordinary voice, the noise-producing instruments are set to work in both ears, with the result that the subject will raise his voice to a shout in an effort to hear what he is saying. It is obvious that if one has total deafness in one ear and the instrument is set going in the good ear the same result will take place, and the patient will raise his voice. This is essentially a test for unilateral destruction of an aural labyrinth.

A rough classification of the subject allows five headings under which one may set down such cases as fall under the observation of otorhinolaryngologists.

1. Anatomic defects are most often congenital, but some occur as the result of the effort to grow up and live in a world full of pitfalls for the struggler who would survive the "slings and arrows of outrageous fortune." Dr. Greene has dealt so fully with the speech incident to such conditions as cleft palate, hare lip and dental malocclusions that it need be mentioned by me only en passant. Diagnosis can often be made by the ear alone. Treatment is, of course, surgical, followed by training in the art of speech. No matter how well the surgical work has been done, or how excellent the healed area looks, training in speech is necessary in every case. Barring this, specialists have not completed their task. Not only do parents have to be advised, but they often have to be commanded, to put the child under a teacher of speech, the selection of whom must be made by the physician and not left to just anybody who happens to be around, for bad or inefficient teaching puts a black mark against every one concerned and may vitiate a child's entire future.

2. The severe colds of childhood, often misnamed "purulent rhinitis," take their toll of hearing, especially when superinduced by diseased tonsils and adenoids, the result of many attacks of "sore throat." In the presence of enlarged, infected adenoids, extension into the eustachian tubes is the rule, with frequent earache, otitis media, mastoid infection or complications resulting therefrom—a march of events that is the same old story to the specialist but a sad, sad story for the growing boy or girl. To be sure, many recover completely, with excellent hearing, but before one discharges such a patient from one's care one ought to be sure that treatment of the nose, throat and ears (including inflation) has done all that is humanly possible to restore the hearing. One ought to be particularly certain that there are no "rests" or foci of lymphoid tissue, which are so often the habitants of

the fossae of Rosenmüller. Such areas are easily destroyed by the engaging finger, and an operation for adenoids is not complete unless one carries out this simple procedure.

Sinusitis in children has been so fully written up by pioneers like Dr. Lee Wallace Dean and the late Dr. Lewis Coffin that one need do little more than mention it. However, there is still plenty of evidence to show that the condition is often overlooked or, if diagnosed, is poorly managed, partly because children under 12 years are so hard to work with, defeating, as they so often do through fear, every effort to help them. Parents, too, often dread the "ordeal" and fail to keep returning for office treatments.

Diplomacy and tact will be tried to the utmost by such patients, and one may have to resort to all kinds of bribery and cajolery to keep them coming. Parents are often difficult, even recalcitrant, and seem to assume that the doctor is merely "trying to run up a bill." This is a good argument for a flat rate arrangement whereby the patient must continue his visits, regardless of the number, until one releases him from that obligation. In the following case the parents had to be educated to their obligation.

A boy 9 years of age was brought in by both parents for examination of his hearing. At 4 years he had undergone tonsillectomy and adenoidectomy, which seemed successful, but for a long time he had recurrent colds; the latest, which was still with him, followed an orgy of wading through pools of water after a hard rain! The story was that he went for months without "ear trouble"; then his ears pained and discharged freely. It was regarded as "just one of those things," and it seems not to have occurred to the parents that an otologist ought to be consulted, until twenty-four hours before, when the left ear discharged *blood!* This caused alarm; it is always fortunate for the patient when a show of blood brings him to a physician, but too often this comes late rather than early in the course of a disease. Here it came early or, at least, early enough. Examination showed purulent discharge from both ears. There was bright red blood on the cotton used to wipe out the left one. Underlying it was a large perforation, plugged by granulation tissue. Hearing in both ears was reduced to about 50 per cent of normal, but one could not be sure what it ought to be when the ears were quiescent. The tonsils had been well removed, but there were definite rests of adenoid tissue around both tubes. When told that the nasopharynx required surgical treatment, the parents demurred. What was the use of going through all that again when it did no good the first time? It required much persuasion and argument to secure permission to send the boy to the hospital. However, this was done; the infected masses were cleaned out, and within a few days the pharynx was healed; the ears were dry, and the hearing was within 10 per cent of normal. If the tendency to colds continues, an effort will be made to secure an autogenous vaccine. Sinal studies (stereograms) will be carried out, and whatever treatment is then indicated will be advised and, it is hoped, carried to a successful completion.

In many cases such early deafness is curable if the otologist can only secure the full cooperation of parents or guardians. This is the

most favorable of all types of obstructive deafness of infectious origin in its response to the efforts of the otologist.

Vocal defects in this classification are commonly associated with colds in the head. There is a lack of nasal resonance. The child is said to talk *through his nose,* which is, of course, a misinterpretation of fact, for he talks *without his nose.* The turbinals are swollen so that they act as a cushion to sound and prevent air from vibrating within the nasal fossae. What one hears is described as "muffled" or "denasalized." Awkward shifts in vocal placement or register take place, but these are only temporary, and they clear up with the subsidence of the conditions which brought them about.

3. Speech defects associated with loss of hearing for certain frequencies are common. The patients usually refer to themselves as "slightly hard of hearing." They do not admit that they are deaf, for the natural tendency is to minimize this fault lest they should be thought stupid. Such losses may be found in the low pitch range, and in this event the voice is strident, hard or muffled and largely uninflected, giving rise to a disagreeable monotone which is often accounted for by saying that a person is "pitch deaf." When such a condition begins in childhood, enunciation of syllables and words is imperfect, and a condition arises which psychologists call "retardation in general language skills." When such partial losses of hearing take place in the high pitch range, even in the zone which lies beyond that generally used in association with speech, difficulties of articulation are constant. Most of them are concerned with consonants, particularly sibilants. From childhood, speech defects persist in the form of "baby talk" or lisping or of general retardation in the development and use of the subtler phases of expression.

Any loss of hearing and speech in this classification calls for something more than merely medical care. There must be, in addition, a pedagogic approach to the problem, and, in my opinion, only teachers of speech who are qualified by training and experience should undertake the education of both hearing and speech. It is useless and wrong to stress either skill at the expense of the other.

4. Profound deafness in children is either congenital or acquired, and in by far the greater number it is acquired. There are, indeed, few congenital deaf-mutes, for careful study will show islands of residual hearing previously unknown to have existed. In 1910 Prof. Alfred Denker, of Erlangen, published a monograph on "Die Anatomie der Taubstummheit" to which contributions were made by Quix and Brouwer, Uffenorde and Schönemann with an integration of their findings by Denker himself. They carefully analyzed in all details the microscopic anatomy of the labyrinth and brain in a deaf mute, but

regarded the case as a "find," a rara avis in the field of pathology. Many who are thought to have been born deaf suffered a severe infection, syphilis or meningitis, in the prespeech period, and the condition was, therefore, set down as congenital.

For otologists the question has, however, only academic interest, since the loss of all serviceable hearing classifies a person as deaf-mute, no matter what the pathologic studies might show. It is much more important to recognize that such conditions have been regarded as hopeless throughout all time and that no one has tried to do much about them until recently. Dr. Max Goldstein, of St. Louis, was a pioneer in undertaking this important task, and to him belongs much of the credit for the present efforts to ameliorate the fate of the profoundly deafened.

The acute infectious diseases of childhood, especially scarlet fever and meningitis, have taken a heavy toll of the labyrinth and of the auditory pathways in the brain as well. Acute suppurative labyrinthitis and meningitis used to be common in childhood. They are less so now because of an increased knowledge of their origin and management and also because means of human communication have so greatly improved that no one lives far from a center where prompt medical care can be obtained. Children are still committed to schools for the deaf but are being cared for by otologists, so that such factors as disease of the tonsils and adenoids, chronic discharge from the ears and other basic elements are removed, giving a chance to the teacher to develop whatever residual hearing may be present. For many years the sign language has been pushed further and further into the discard. In its place have come lip reading and the use of electrical amplification properly based on the results of audiometric studies. Hearing aids are still imperfect, but they have improved marvelously during the past five years because engineers, physicists, psychologists and otologists have combined their efforts to solve the problem of making the deafened hear. That problem also involves the teaching of speech, urging the pupil to try to imitate the sounds he hears. Fortunately, speech centers and speech organs are usually intact after the smoke of battle has cleared and the fight for life has been won by the little patient. The effort to hear followed up by the effort to speak is, unfortunately, fatiguing. It makes great demands on the nervous system, and only a little can be accomplished at one sitting. Moreover, once the pupil is released from a classroom he throws aside his hearing aid and reverts to signing, since that is along the line of least effort to establish communication with others.

Then too, the pupil shrinks from normal persons as much as he can, preferring the society of fellow sufferers. This delays satisfactory adjustment, vitiates results and makes the teacher's task much harder than it ought to be. All that it is intended to point out in this little

essay is the necessity for the otologist to cooperate with teachers, manufacturers of hearing aids, physicists and electrical and radio engineers in order that he may do his part in trying to restore the deafened person to a better place in society, thus helping him to earn a living and to remove a heavy burden from taxpayers, philanthropists and the state. It can be done, and it is being done in many cities and centers within the United States. England, however, has gone ahead much faster in her work with the deafened and has made excellent contributions through studies in the public schools and in laboratories of all kinds.

If deafness has occurred after speech has been well developed, vocal defects are not so marked, but there is often nasality, poor inflection and awkward shifts between guttural and high-pitched, strident tones and always that dread monotone so characteristic of deaf-mutism.

5. Profound deafness in adults brings about changed quality of speech and intensity. Nerve deafness is always associated with a peculiar voice. Since the patient cannot hear his own voice he either shouts or mumbles, and in either case it is difficult to understand him until one has become accustomed to his method of production. The effort is made to hear one's own voice, with a result that is harsh and unpleasing to the listener, who soon tires of trying to carry on a conversation. Too often the deafened person insists on doing all the talking so that he will not be bothered with questions which he can neither hear nor understand. With conduction deafness, as distinguished from nerve deafness, voices are generally faint, mumbling and indistinct.

The varieties of speech are as numerous as the sands of the sea. Every one possesses the inherent ability to vary the voice in pitch, quality and intensity. One can so change one's voice as to make it absolutely unrecognizable when one is out of sight, even by the friends who know one best. The voice one happens to claim as one's own is the result of a settling down after puberty and it is high, medium or low according to individual characteristics. The spoken voice is, however, a poor index of virility or effeminacy. One of the most masculine men I ever knew had a miserable, high-pitched, disagreeable voice, which carried no conviction, chiefly because no one ever listened for the thought conveyed by it. The listener was aware only of the mechanical effect on his auditory mechanism and not at all, or rarely, of the message the speaker was attempting to convey. And the pity is that that man need not have gone through life with such a voice. It could have been brought down by a skilled teacher to almost any desired level and kept there through paying attention to the quality and the method of production. It might never have been beautiful, but it could have been made effective.

It is not true that a normally hearing and speaking person has no idea whatsoever of the kind of voice he has. Every one knows whether he speaks in a high or low voice; he ordinarily knows when he shouts (save in anger, when one is almost entirely objective) and when he speaks in a subdued manner. But in truth persons seldom pay much or any attention to their method of speech. They indulge in a badly produced, badly modulated voice, which grates and rasps on listeners and defeat their social aims without realizing the fact. Elegant speech is such a rarity that one is almost shocked when one hears it and is led to exclaim, "What a beautiful voice he has!" But all hearing persons, if they were as interested as they really ought to be, might well have a pleasing speaking voice if only they were willing to "listen in" on themselves and correct obvious faults in production.

Reading aloud, which is a lost art to most, is one of the best ways to observe one's own way of speaking. In fact, the reader may not understand a word of the thought being conveyed if he is intent on the manner of its conveyance. A year or two ago, I thought it would be a rather nice gesture to make a record of Kipling's "If" and Henry van Dyke's "America for Me" and pass it on to my growing son as a Christmas present. When the thing was done and the opening of gifts was in order, I proudly brought forth the disks, put them on the playing machine and watched for the result. At the end of the first piece, the boy turned and said: "Who on earth was that?" Well, the result was not flattering! I suppose that in my great desire to produce a good and faithful record I overstepped and made my voice sound quite different from its usual style.

Some preachers, whose noble work in life it is to expound the scriptures and induce an audience to think, for a time at least, on higher things, have little or no accurate idea of delivery. They stand on tiptoe and scream or bawl at their hearers in a way that is little short of ludicrous, much as one should like to be kind and tolerant of them. They have not been taught, or, at least, they have not learned, how to speak effectively. This is true of many sects and divisions of the Christian church. In all fairness, however, one may largely absolve the clergy of the Church of England, many Catholic priests and the rabbis who speak from the radio. Most of them have fine, well modulated, resonant voices and carry a feeling of reverence and conviction to their hearers. The Lord's Prayer and the Sermon on the Mount may well be ruined for a listener by the speech pattern of the ecclesiast, who seems well convinced that he is uttering great teachings in a thoroughly legitimate manner. Most voices, let it be said, which are heard via the radio are well produced and effective. Somehow the radio has a way of weeding out bad voices save when the speaker is strictly on

commercial programs. The sustaining programs usually have fine, polished and thoroughly "listenable" voices.

As for university lecturers, it is a bit startling to find that many of them have had no training in speech or hearing whatsoever. They are employed on evidences of their intellectuality, usually based on written theses or published books, plus sufficient academic "pull." Many of them know little and care less about their manner of delivery. It is sufficient to cast their pearls of wisdom in any manner that pleases them. If the student can pick them up, very well, but if not it is his fault and not the fault of the speaker. But all that is going to be changed, and better speech is likely some day to come into its own even in the seats of higher learning.

At Syracuse University the general health examination of entering students now includes audiometric measurements and speech tests. The student health service and the school of speech have combined in an attempt to disclose each student's actual difficulties in hearing and speech. Surprising results are being recorded. For example, one student insisted that he was not very deaf. An audiogram showed more than 80 per cent loss of hearing in each ear! Another, who complained that he had a great deal of difficulty in hearing lecturers and who had been undergoing corrective treatment for dyslalia for some three years, was found to have a loss of hearing of only 12 per cent in one ear and 15 per cent in the other!

Let it be hoped that other universities will imitate the good example set by Syracuse.

Voices convey to a listener something by which he judges mentality and strength or weakness of personality. One may be misjudged in a downward direction; seldom is one rated upward if quite unworthy of good opinion. Few succeed in life in mastering men if they have voices that grate on the nerves or are otherwise deficient to the listening ear. A notable exception to this rule was the first President Roosevelt. He was possessed of a shrill, high-sounding, penetrating voice, but somehow the message he conveyed was all absorbing, so that no one paid much attention to the way in which it was delivered. But then "T. R." was a law unto himself in every other way as well. He set a pace in all things which few men in the world, past or present, have been able to emulate successfully.

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A SIMPLE TREATMENT FOR DEFECTS OF THE SINGING AND OF THE SPEAKING VOICE

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It is not proposed in the discussion of this subject to describe the various methods of treating defects of the singing and of the speaking voice employed by the laryngologist, who is devoting much time and thought to this branch of his specialty. It is my present purpose to discuss a simple method of treatment to be applied not only to defects of the singing, but also to defects of the speaking, voice, encountered in daily practice. Public speakers, teachers, actors and preachers often consult the laryngologist for a defect of the speaking voice, but there is also a vast number of patients who misuse the voice and whose defect remains unrecognized and improperly treated. I refer to saleswomen, persons reading to the blind, persons attending cocktail parties or teas, who use their voices in the presence of a loud and constant noise, and those who speak loudly to the deaf. These patients may complain of vague pains in the neck and of hoarseness caused by mild paresis of the internal tensor muscles—in other words, acute vocal fatigue—and are often treated for laryngitis instead of for phonasthenia.

It has struck me forcibly that a singer who has been treated by electric phonic compensation for acute phonasthenia in order that he or she may be able to fill an allotted engagement is unable, in the majority of instances, to receive the same treatment by a laryngologist in another city, when on concert tour. There is certainly no good reason for such a circumstance, as the treatment is simple and in acute conditions effective and should be known by every laryngologist. To bring this subject to the attention of members of the medical profession is the sole purpose of this paper.

If a singer is physically and mentally fit and if he sings in a manner physiologically correct, there need be no concern about the condition of the larynx, provided he does not sing to excess. It is perfectly apparent, however, that, should there be some systemic derangement, the delicate mechanism of the vocal cords becomes easily disturbed. The principal systemic causes of vocal fatigue are anemia, pulmonary tuberculosis, heart lesions, chronic toxemia, pregnancy, menstruation and

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endocrine dysfunction. The last named, in the form of hypothyroidism, is a frequent cause and must be given the attention it deserves.

Local causes, such as chronic laryngitis, singer's nodules, dilated blood vessels of the vocal cords, enlarged lingual tonsils, infected faucial tonsils and adenoids, abnormalities of the nose causing obstruction and disease of the paranasal sinuses, may be contributing factors.

The chief cause is usually misuse of the voice. This, according to Flatau,[1] may be quantitative or qualitative. The first applies to the use of the voice for too long a time and the second to a faulty method. Qualitative factors may be summarized as follows: forced fixation of the larynx, coup de glotte—i. e., rapid approach of the vocal cords in reaching a note—improper use of the lips, tongue and lower jaw, insufficient and forced vocalization, improper breathing, lack of relaxation and the use of the voice in the presence of an acute infection of the upper part of the respiratory tract.

One of the first symptoms complained of by the patient is a feeling of fatigue in and about the larynx. A tired feeling after great exertion is not unusual and is not to be looked on as one of the symptoms of phonasthenia. The tired feeling which is of concern to the laryngologist is the one which occurs earlier, as a rule about fifteen minutes after one begins to sing or speak. When pain becomes a prominent symptom, it may localize itself in the larynx or in the occipital region and may radiate to the shoulders and sternum in some cases.

Parasthesia is often present and is usually disturbing to the patient. The parasthesia is not well localized. It may be in the postnasal space, nose or larynx and may occur in the form of pressure, dryness, burning, itching or a sensation as though a foreign body were present, or "globus hystericus."

The objective symptoms are those one hears and sees. When intensity is disturbed, the patient cannot sing or speak in a loud tone, and the voice is changed in character. A disturbance of extensity means that the singer has lost some of the notes or has a tremolo. The most frequent symptom is detonation—in other words, the patient sings flat—and he is many times not aware of this defect.

The laryngeal examination may reveal changes in the form and disturbances in the movements of the vocal cords. The following changes may be seen: singer's nodules, glandular cysts, irregularity of the free edge of the cords, thickening of the interarytenoid space and changes in color. Singers do not have a normal color of the cords. There is usually a roselike tint, more pronounced after singing, which is in reality an occupational hyperemia—in a way a physiologic change.

1. Flatau, T. S., in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1929, vol. 5, p. 1301.

The disturbance of movements of the vocal cords may be twofold; first, there may be a lack of movement, with incomplete closure, and, if the paresis is mild, there may be some difficulty in recognizing its presence. Second, fortunately rarely, the adductor muscles may be so strong that the arytenoids override and asymmetry results.

It must always be remembered that the larynx is sensitive and that, as the adductor and abductor muscles do most of the work, misuse of the mechanism will result disastrously. Circulatory and secretory disturbances may occur in the larynx, and a catarrhal inflammation of the vocal cords, with hypersecretion of mucus, is the result. The catarrhal inflammation is usually confined to the larynx itself, the upper respiratory regions being unaffected. On examining the larynx one often sees a sticky plug of mucus, hard to dislodge, lying on the vocal cord. If phonasthenia has been present for a considerable time, dilatation of the blood vessels of the vocal cords with thickening of the free edges may follow. If there is marked thickening of the cords, paresis of the internal tensor muscles may easily be overlooked.

It is well known that each singing teacher has a distinct method of his own, when in reality there is only one method of teaching the art of singing and that is the one by which it is done in a physiologically correct manner. Furthermore, it happens in many instances that all students are instructed in a like manner, which is undoubtedly a mistake, since individual attention should be given to the faults of each singer and an attempt should be made to correct these faults along physiologic lines. One of the details most frequently overlooked—and one which is difficult to master—is diaphragmatic breathing with proper control of the abdominal muscles. Vocal fatigue will continue to be frequent so long as singing teachers continue to use their special method on every pupil, fail to individualize instruction for each voice, allow the pupil to sing loud in order to overcome the beginning paresis of the vocal cords, permit him to sing in the presence of an acute cold and suggest that he should practice hours at a time in order to develop and strengthen the voice. A voice properly trained may continue to functionate in a splendid manner until, as the result of excessive singing or the development of a faulty technic, the first symptoms of phonasthenia, such as detonation and a slight tremolo, make their appearance. I am rather inclined to the opinion that many voices could be saved if a functional examination of the larynx by means of phonic compensation were made at stated intervals. If this were done, vocal fatigue would be detected in its early stage and, with a few treatments, easily corrected.

I shall endeavor, in a brief way, to discuss the salient points pertaining to the examination of the phonasthenic patient. Indirect laryngoscopic examination has its disadvantages, as the parts are dis-

turbed when the tongue is protruded. However, fine changes on the vocal cords may be more easily recognized by using a magnifying laryngeal mirror. The character of the breathing should, if possible, be ascertained. To determine the length of expiration, the patient is asked to take a deep breath and at the time of exhalation to hum the consonant "m." Many times it will be noticed that a large part of the expiratory current of air is rapidly expended. This is a faulty method of breathing and shows lack of control of the abdominal muscles and improper diaphragmatic action. It is needless to add that if this type of breathing is continued for any length of time vocal fatigue will follow. By placing a band in the region of the diaphragm and asking the patient to expand the band by taking a deep breath and allow for a slow retraction of the abdominal muscles while humming the consonant "m," proper breathing is easily taught. This humming exercise is of great benefit also to public speakers who are inclined to use a high-pitched voice, thereby elevating the larynx, instead of keeping it in a normal position while speaking.

There are two tests which may be used when a singer's chief complaint is lack of resonance; namely, the "ah ee" test of Gutzman [2] and the cocaine test. In the "ah ee" test the singer enunciates "ah ee" and at the same time opens and closes the anterior nares repeatedly by compressing them with the fingers. In rhinolalia aperta an increase in the nasality occurs, and in rhinolalia clausa there is no difference in the tone of the voice. A nasal obstruction may, therefore, be the cause of the lack of resonance if no difference in the tone is heard. In using the cocaine test, a small quantity of a 2 per cent solution of cocaine is sprayed into the nose, and the resonance of the voice is tested by having the patient vocalize before and after its use. If the resonance is improved, a nasal operation may be indicated. It should be emphasized, however, that the removal of a large amount of tissue from a singer's nose may produce a marked change in the voice of a character not pleasing to the ear. In many instances, conservative measures for the reduction of edema of the inferior turbinate, such as application of trichloroacetic acid or electrocoagulation will suffice.

The question often arises, When should tonsillectomy be performed on a singer? The decision is easily reached if it can be shown that chronic infection of the tonsils is undermining the general health of the singer and that the infection is interfering with proper movements of the soft palate. When it has been decided that a singer needs tonsillectomy, it is well to establish before the operation is undertaken whether there is a defect of the singing voice. The tonsillectomy can, therefore, not be blamed for a detonation discovered before the operation if it persists after healing is complete. The operation itself should be done

2. Zumsteeg: Ueber Phonasthenie, Arch. f. Laryng. u. Rhin. **24**:1, 1910.

with the greatest care, so that injury to the soft palate is reduced to a minimum. The postoperative period should be rendered as free from pain as is possible by making free use of powders containing acetophenetidin, acetylsalicylic acid and codeine. Intense pain after the operation is followed by severe reaction, and the latter, in turn, is productive of scarring and retraction of the soft palate, which is undesirable in a singer. The patient should not take a prolonged rest after the operation but should, instead, begin mild vocal exercises at the end of two weeks, vocalizing more each day, so that in a month the full voice may again be used. It is known that some patients complain of dryness of the throat following tonsillectomy. When this occurs in the singer, it may, as shown by Stern,[3] be the result of chronic stage fright, for it is known that the mouth and pharynx are dry when one is undergoing a severe nervous strain.

It is easy to understand why detonation is a frequent defect of the singing voice when one remembers the delicate structure of the laryngeal muscles and the abuse which is imparted to them when their mechanism is disturbed by improper singing and speaking. It is unfortunate that many times a singer does not know that a flat note is being sung and does not realize that there is anything wrong until a distinct break takes place. In such cases one is usually dealing with advanced phonasthenia. Generally speaking, however, there is a loss of clearness of tone, and the experienced singer recognizes it on attempting to vocalize. How is the laryngologist to know when false notes are being sung? This is done by the use of the chromatic pitch pipe. Beginning with a low note, the pitch pipe is blown, and the patient is asked to sing softly (pianissimo) the vowel "o," preferably "lo," each time a note is emitted from the pipe. If possible, the entire chromatic scale is tested, and the laryngologist makes note of the following:

1. Clearness of tone.
2. False notes.
3. Break in the voice.
4. Ability of the patient to hold a given note.

It is perfectly apparent that the foregoing method of examining the singer can readily be used by the laryngologist who has a musical ear. The question arises, how is one to proceed if the opposite is true? In this instance, the statement of the patient regarding the presence of a vocal defect is sufficient reason for proceeding with the treatment of the voice.

It is my intention in discussing treatment of phonasthenia to speak only of a simple method, the use of which has been found satisfactory

3. Stern: Die Tonsillectomie in der Sängerpraxis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **15**:193, 1926.

in the majority of instances. I refer to the application of a mild faradic current to the exterior of the larynx during singing and speaking. It is a known physiologic fact that when a note is sung and a mild faradic current is applied to the exterior of the larynx, the pitch will rise. It therefore follows that if a singer is singing flat the faradic current will compensate the detonation and will cause the pitch to rise and the note to be sung in a normal, clear tone. This is termed electric phonic compensation. The strength of the faradic current should be such that the patient barely feels it. There must be no pain or muscular contraction. If this occurs, the opposite takes place—the note breaks and phonic collapse may result.

Imhofer [4] rightly emphasized the fact that by the use of electric phonic compensation one can determine whether a patient is able to fill a singing engagement. If, while one is using electric phonic compensation, each note rises in pitch and notes which were sung flat once more become normal and clear in tone, the laryngologist need not hesitate to advise that the singer keep his or her engagement. In fact, the outcome of the functional faradic compensation tests is far more important than the appearance of the larynx when a decision is to be reached as to the advisability of allowing a singer to perform. It is obvious that the exception to this rule is the presence of acute laryngitis.

In applying the treatment, the laryngeal electrode, moistened with saline solution, is placed on the neck about a centimeter from the thyroid cartilage. Beginning with a low note of the scale, the tone is given by the pitch pipe, and the singer sings the note in pianissimo by using the word "lo." The faradic current is not applied until the patient has begun to sing and is continued so long as the note is being held. Four or five applications of the faradic current are usually made to each note before proceeding with the next note higher in the scale. It is self evident that if a tone rises on application of the electric current further stimulation on that note is unnecessary. Detonation usually occurs—especially in sopranos—at D and E of the middle register, and in cases of acute vocal fatigue is readily compensated. By having the patient vocalize before and after the treatment, the effectiveness of phonic compensation may easily be determined. When treatment for vocal fatigue is used on patients who are not singers, the faradic current is applied while the patient is speaking, care being taken that proper breathing and tonal placement are being used.

It is often suggested to singers who have a defect of the voice that they take a prolonged rest. This is good advice if singer's nodules are the cause of the trouble, as nodules have been known to disappear

4. Imhofer, R.: Die Ermüdung der Stimme (Phonasthenie), Würzburg, Curt Kabitzsch, 1913, p. 83.

after three or four months' rest of the voice. With phonasthenia, however, it is advisable to have the patient continue singing in a milder degree, if possible, and to institute treatment by means of phonic compensation. The presence of a plug of mucus may necessitate irrigation of the larynx with a physiologic solution of sodium chloride or, better still, with the following solution:

Alcohol	30.0
Oil of peppermint	3.0
Distilled water	150.0

Filter.

It is worth mentioning, however, that local treatment alone, without phonic compensation, is usually of no avail against catarrhal affections of the larynx resulting from misuse of the voice.

It is not within the scope of this paper to describe other methods of phonic compensation. It was my desire to explain a simple method, one which requires no special training in the treatment of vocal defects and one which can readily be used. When once understood, this method of treatment of defects of the voice in singers, public speakers and other patients becomes simple and practical, and because of its simplicity it should be a routine measure of every laryngologist.

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THE VOICE AFTER DIRECT LARYNGOSCOPIC OPERATIONS, LARYNGOFISSURE AND LARYNGECTOMY

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Voice is the chief consideration in the treatment of most benign laryngeal conditions. In malignant conditions voice must obviously be relegated to second place, because the first and most urgent indication is to cure the cancer. It is necessary to explain this to the patient and his family, so that they may readjust their attitude and accept whatever vocal impairment may be entailed in the procedure required to obtain the proper prospect of cure. However, the fact remains that one of the things that the patient is most anxious to know before consenting to the necessary operation is whether he will lose his voice. Many people believe that removal of the vocal cords will make it impossible for them to talk, and the majority of those to whom total laryngectomy is advised are skeptical when assured that they will soon learn a new way of talking after the larynx is removed. It is explained that voice is relatively unimportant as compared with the cure of the cancer, which is inevitably fatal if untreated, but it is comforting to the patient and conducive to his fit condition for operation, as well as a consolation to his family, to know that he will not be doomed to use sign language or a pad and pencil the rest of his life to express himself.

It seemed to me that it would be interesting and helpful to conduct a special follow-up on patients who have had the various laryngeal operations, by sending out questionnaires regarding postoperative vocal development. Of the first group of patients, those to whom direct laryngoscopic treatment was given, only one small series was questioned, patients for whom nodules of the vocal cords ("singers' nodes") had been removed. Of the second, those who had had more radical operations, for the extirpation of cancer, all living patients operated on since 1929 were sent questionnaires. Replies were received from 51 patients who had had one or both cords removed by laryngofissure and from 30 patients on whom total laryngectomy had been performed.

From Temple University Hospital.

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THE VOICE AFTER REMOVAL OF NODULES OF THE VOCAL CORDS

Direct laryngoscopic procedures [1] are done for the treatment of papilloma, organizing hematoma (benign tumor of inflammatory origin), polypoid corditis, eversion of the ventricles, nodules of the vocal cords and many other conditions, but only the group treated for nodules of the vocal cords was chosen for questioning. This group was chosen for two reasons: first, because it was relatively small and, second, because there has been some difference of opinion as to whether such nodules should be removed and as to the permanence of the result obtainable by removal.

After the removal of papillomas or other benign true tumors, unless the underlying normal tissues have been damaged by the present or some past procedure, by secondary inflammation or by excessive strain, the voice will be restored. One interesting observation that I made recently in a case of chronic papilloma in an adult might be mentioned here. Whenever the papilloma recurred slightly a tendency to phonate with the ventricular bands was noted; the bands appeared to attempt to reenforce the true cords, but the result was not satisfactory. Removal of even a small amount of papillomatous tissue from the margin of the true cords was sufficient to restore normal cordal function and a good tone of voice. The patient is a lawyer, and his voice is important to him. The phonation with the ventricular bands would probably be classified as "vicarious" in this case, though the mechanism came into play so readily that it might be considered "usurpative." [2]

The removal of "organizing hematomas" should be followed by perfect restoration of voice unless chronic laryngitis is associated or the normal tissues are damaged by unskilled use of the forceps. My colleagues and I have always advised scalping off benign tumors superficially without making any attempt to deal radically with the base. We prefer to leave the cordal margin slightly convex rather than concave. Not infrequently it is best to remove the tumor in two or three stages, but in the majority of cases it can all be removed at one.

In polypoid corditis and eversion of the ventricles, an amazing improvement in voice can be obtained by carefully scalping off the excess tissue, first from one side and later from the other side of the larynx, but it is always worthwhile to prescribe for patients with these con-

1. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937; Cancer of the Larynx, J. A. M. A. **111**:1986 (Nov. 26) 1938. Jackson, C. L.: The Indications for Direct Laryngoscopy, Tr. Am. Laryng., Rhin. & Otol. Soc., 1939, to be published; Ann. Otol., Rhin. & Laryng., 1939, to be published.

2. Jackson, C., and Jackson, C. L.: Dysphonia Plicae Ventricularis: Phonation with the Ventricular Bands, Arch. Otolaryng. **21**:157 (Feb.) 1935.

ditions a preoperative period of about the same, or somewhat longer, duration, during which they should observe the greatest moderation in talking, smoking and drinking.

As already stated, there is some difference of opinion as to whether or not true nodules of the vocal cords should be removed, but it has been my experience that they rarely disappear spontaneously, whereas their removal by direct laryngoscopic procedure gives excellent vocal results and they do not, as a rule, recur. However, Ridpath [3] was probably correct in saying that if the nodules are not too well established they may disappear, provided faulty voice placement is corrected. Voorhees [4] stated the indications for treatment accurately in his book on "Hygiene of the Voice." Others who have discussed the subject are Imhofer,[5] Sokolowsky [6] and Prévot.[7]

Questionnaires were sent to a series of patients who had had nodules of the vocal cords removed, and at the present time 13 have replied. All but 2 of these stated that their voices were "good"; the other 2 said "fair." These patients had been hoarse for periods varying from three months to twelve years prior to operation. For the 10 patients who had been hoarse for a year or more, the average duration of hoarseness was over four years.

Despite the good results obtained in this series, I am inclined not to advise immediate removal of the nodule in the case of an operatic singer. I believe that in such persons a trial of vocal rest for two months, followed by gradual resumption of singing, is indicated, care being taken when singing is resumed to improve the voice placement and the manner of singing, in cooperation with the singing teacher. If the nodule persists after this program has been carried out removal may be again considered.

THE VOICE AFTER LARYNGOFISSURE

Patients who have had one or both true cords removed by laryngofissure practically always develop a useful voice, but its quality, pitch and carrying power vary greatly. Cicatricial "adventitious" cords develop in most cases, and in the patients with good voices, generally either one good cord remains, and a good "adventitious" cord has devel-

3. Ridpath, R. F.: Voice Production, with Suggestions for Treating Hoarseness and Loss of Voice in Singers, Atlantic M. J. **26**:831 (Sept.) 1923.

4. Voorhees, I. W.: Hygiene of the Voice, New York, The Macmillan Company, 1923.

5. Imhofer, R.: Ueber Sängerknötchen, Ztschr. f. Laryng., Rhin., Otol. **12**: 79, 1923.

6. Sokolowsky: Sängerknötchen und Stimmstörung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:246, 1925.

7. Prévot, M.: Le nodule vocal chez le chanteur, J. de méd. de Lyon **7**:411, 1926.

TABLE 1.—*The Voice After Removal of Nodules on the Vocal Cords*

Patient	Sex	Age	Date of Direct Laryngoscopic Procedure	How Long Were You Hoarse Before Treatment?	Is Your Voice Good? Fair? Poor?	Do You Address Groups?	Did You Sing?	Are You Able to Sing Now?	Comment
1. M. A. A.	F	32	11/29/37	About 1 yr.	Good	No	No	No	"I wish to express again my sincere thanks to you, as I consider the operation very successful."
2. A. B.	F	35	1/19/39	7 mo.	Good	No	No	No	
3. H. C.	F	42	6/17/35	2 yr.	Good	No	No	No	"I have had no trouble whatever with my voice since your operation."
4. M. A. D.	F	44	11/28/38 12/12/38	About 12 yr.	Good	No	No	No	"My voice was improved 100 per cent, and I consider the operation a complete success."
5. C. G.	F	42	7/13/36 7/23/36	10 yr.	Good	Yes	Yes	No	"I find that my voice tires more easily than before I became hoarse. I had a strong vibrant voice before the hoarseness. Now my voice, although clear, is not as strong."
6. S. J.	F	17	3/ 9/39	3 mo.	Good	Yes	Only at school	A little	"When the weather is bad my throat is irritated."
7. C. J.	F	39	11/22/34	About 3 mo., continuously	Good	No	No	No	"As my record probably shows, I became hoarse when about 21 years old, while teaching school. I had recurring attacks several times yearly, even after giving up teaching. The condition was always worse in bad, misty or foggy weather or when I was under mental or physical strain. Since operation I have had no attacks of hoarseness, and my voice is not affected by the weather, colds or my general state of health. I am much pleased with the results of the operation. My voice has a husky, sometimes slightly strained, tone, but I do not get hoarse or lose my voice as I did for so many years."
8. B. K.	F	31	6/18/31	About 2 yr.	Good	No, but I teach	No	No	
9. A. S.	F	43	9/12/29	2 to 3 yr.	Good	No	No	No	"I have hoarseness only when I do excessive talking or use the telephone."
10. E. F. S.	M	59	10/12/36	About 1 yr.	Good	No	No	No	
11. J. J. T.	M	46	11/ 5/36	Approximately 14 mo.	Good	Occasionally	No	No	"If I talk too long at one time my throat seems to get dry and somewhat fatigued."
12. M. T.	F	39	11/15/34	1 yr.	Fair	No	No	No	"I am happy to state that the operation helped me and that I feel much better."
13. O. P. G.	F	35	9/27/37	For years	Good	No	No	No	"The only time I ever notice any hoarseness is when I am overtired and I have to talk over the noise of music or there is a large group of people and a lot of noise."

oped, with which it can approximate and vibrate, or two good "adventitious" cords have formed to replace two cords that have been removed. Another mechanism by which a good voice can be produced is phonation with the ventricular bands,[2] which is to be regarded as undesirable when it is "usurpative" but which may afford an excellent and efficient substitute for phonation with the true cords when one or both cords have been removed. One of the factors that is of importance in determining the character of the voice is the degree of motility, and this in turn generally depends in some measure on how much of the arytenoid was removed and how severe the inflammatory reaction was in the cricoarytenoid joint. The presence of a granuloma may interfere seriously with the production of a good tone, and such tissue should be removed by direct laryngoscopic procedure if it does not shrink and disappear in a month or two after operation. Sometimes several removals are necessary. In still other cases the voice is impaired by a cicatricial web across the anterior commissure.

Of 51 patients submitted to laryngofissure in the past ten years who have answered the questionnaire at the present time, 43 replied in the affirmative to the question "Have you developed a useful voice since your operation?" To the question "Is you voice good, fair or poor?" 8 replied "good," 37 "fair" and 6 "poor." Persons with a great variety of occupations were included in the series: physicians, lawyers, bankers, salesmen, teachers, editors, machinists, clerks, electricians, merchants, farmers, business executives and housewives. Thirty-seven of the patients stated that they had been able to resume the same work that they had done prior to operation. Forty-three were able to use the telephone, and 10 stated that they were able to address groups of people, some of them qualifying the statement with the word "small." Sixteen patients could sing before operation, but only 3 asserted that they could do any singing since laryngofissure.

I am now at work on a follow-up study of the postoperative appearances in the entire series, with a view to determining, if possible, what elements are participating in the voice developed in each particular case.

SPEECH AFTER LARYNGECTOMY

The patient who has had his larynx removed or is about to have it removed is apt to be depressed at the thought that he may never be able to talk again, but he will be cheered at once if one explains to him that the larynx is only one part of the speech mechanism, that it provides a column of air (coming up from the lungs) and a pair of vibrators (the vocal cords) but that it is the "molds of speech" (such as the tongue, lips, cheeks and palate) which form the words. Then one

TABLE 2.—*The Voice After Laryngofissure*

Patient	Sex	Age	Date of Operation	Have You Developed a Useful Voice?	Is Your Voice Good? Fair? Poor?	What Was Your Occupation?	Were You Able to Resume the Same Work?	Do You Talk on the Telephone?	Do You Address Groups?	Did You Sing?	Are You Able to Sing Since Operation?	Comment
1. A. W. B.	F	40	10/27/36	Yes	Fair	Housewife	Yes	Yes	No	Yes	No	"The strength of my voice depends on general physical conditions."
2. M. B.	M	60	3/10/36	Yes	Fair	Carpenter	Pensioned	Occasionally	No	No	No	"My voice is a little above a whisper."
3. J. E. B.	M	62	1/ 9/34	Yes	Good	Executive and part time salesman	Yes	Yes	No	No	No	"I have gained weight (198 lb. [90 Kg.])."
4. S. F.	M	53	12/15/33	Yes	Fair	Hebrew teacher	No	No	No	Yes	No	"I am still unable to breathe freely."
5. J. G.	M	51	10/30/34	Yes	Fair	Farmer	Yes	Yes	No	Yes	No	"My voice is slightly low."
6. L. G.	M	49	7/25/33	Fair	Fair	Drill press operator	Yes, but not working now	Yes	No	Yes	No	
7. P. L. J.	M	63	4/16/38	Yes	Fair	Retired medical officer, U. S. A., now director of hospital	Yes	Some	No	No	No	
8. G. U. H.	M	66	3/13/33	Not very good	Poor	Editor-publisher	Yes	Yes	No	Not much	No	"My voice has not shown improvement in the past several years."
9. G. K.	M	59	9/25/37	Yes	Fair	Salesman	No	No	No	No	No	"I have gained 11 lb. (5 Kg.)."
10. F. S. L.	M	62	9/27/35	Yes	Fair	Clerical worker	Yes	Yes	No	Yes	No	
11. R. W. Mc.	M	59	7/28/37	Yes	Good	Lawyer	Yes	Yes	In court work	No	No	"I have argued several cases and tried several cases with no ill effects. My voice varies; old friends say it is normal."
12. W. F. J.	M	53	11/ 5/38	Yes	Fair	Executive	Yes	Yes	No	No	No	
13. B. M.	F	58	1/ 7/31	Yes	Good	Government clerk	Yes	Yes	No	Yes	No	"My voice grows continually stronger."
14. C. M.	M	70	3/27/35	No	Poor	Lawyer	To a limited extent	Yes, whisper	No	No	No	"I use a whispering voice, which carries very well."
15. A. K. O.	M	58	12/13/30	Yes, in a quiet room	Fair	Assistant vice president of trust company	No	Yes	Occasionally, small groups (20 to 30)	Yes	No	
16. F. T. O.	M	61	10/28/36	Yes	Fair	Executive in charge of sales department	Yes	Yes, with special microphone attachment	No	No	No	"While the voice has improved constantly during the past 2 years, it has not reached a point where it is possible to carry on business conversations except in a quiet office. I have been in good health and work as hard as ever."
17. A. N. P.	M	57	10/27/37	Yes	Fair	Public accountant	Yes	Yes	No	Yes	No	
18. G. A. S.	M	73	3/30/35	Yes	Fair	County appraiser	Yes	Yes	No	No	No	"When I am talking on the telephone and have to repeat, it makes me nervous and short of breath. My voice is always hoarse, as though I had a bad cold."
19. J. G. S.	M	68	7/ 3/33	Yes	Fair	Executive	Yes	Yes	No	Yes	Able to carry a	"I use my voice considerably during business hours, especially for dictation, but conserve

short of breath. My voice is always hoarse, as though I had a bad cold."

19. J. G. S.	M	68	7/ 3/33	Yes	Fair	Executive	Yes	Yes	No	Yes	Able to carry a tune, but volume is limited	"I use my voice considerably during business hours, especially for dictation, but conserve my voice during the evening."
20. J. B. S.	M	45	2/18/30	Yes	Fair	Machinist	Yes	Yes, with effort	No	No	No	
21. T. B. W.	M	58	8/ 6/37	No	Poor	Farmer	Yes	Yes	No	No	No	"I can talk to a small group of people in a small quiet room, where a whisper can be heard."
22. H. W.	F	56	9/28/34	Yes	Good	Housewife	Yes	Yes	No	Yes	Yes	"I think the operation was successful in my case. I am satisfied with my voice."
23. E. W.	M	77	7/21/37	Yes	Fair	Sexton	Yes	No	No	Yes	No	"I can talk on the telephone if necessary but have never had occasion to do so since the operation. My voice is not strong enough to address groups of people."
24. M. B.	F	51	2/20/37	Yes	Fair	Housewife	Yes	Yes	No	Yes	No	"My voice is still rough and husky but gradually getting stronger."
25. E. A. H.	M	63	11/ 7/33	Yes	Fair	Electrician	Yes	Yes	No	No	No	
26. A. T. H.	M	76	9/ 4/36	No	Fair	Merchant	No	Yes	No	No	No	"I do not answer the telephone if I can get out of it. My voice is not strong but much better than a whisper. I think it is gradually improving."
27. H. C. H.	M	45	8/11/37	Yes	Fair	Clerk	No	No	No	No	No	"I did much talking and dictation and telephone work before operation. Now I do work which does not necessitate much talking."
28. P. H.	M	60	11/ 8/32	Yes	Fair	Manager	Yes	Yes	Small groups	I thought so	No	
29. M. B.	M	62	3/28/32	Yes	Poor	Manufacturer	Retired	Yes Yes	Yes, small	No	No	
30. L. L. D.	M	59	4/10/33	Yes	Fair	Colonel, U. S. Army	Yes, until retirement, in 1937	Yes, without trouble, long distance and local	No, except socially, in small room, in general conversation	No	No	"My voice enables me to get along well among strangers when traveling and among friends with whom I am associated. I cannot call to persons at a distance and have trouble in making myself heard by those in another room. Most persons do not notice my voice, although strangers now and then comment on my apparent hoarseness and ask about my cold."
31. C. H.	F	63	4/17/36	Yes, but low	Fair	Housewife	Yes	Yes	Small, in quiet room	No	No	"I have noticed a marked improvement in the last 2 months. I cannot talk over any sort of din or noise. I am in perfect health."
32. G. E. L.	M	53	5/ 8/30	Yes	Fair	Manufacturer	Yes	Yes	At small meetings	No	No	
33. R. N.	M	74	4/ 4/33	Yes	Fair	Manufacturer	Retired now but worked a while after operation	No, cannot be understood; voice is too low	No	No	No	"I am able to talk plainly to people in a room but cannot call or shout. There is mucus in my throat which I was not troubled with previously."
34. W. S.	M	52	5/21/34	Yes	Fair	Farmer	Yes	Have tried, but voice is too weak to be heard	No	No	No	"I cannot talk loud but can whisper distinctly. If I try to talk where two or three are talking my throat gets tired."

Table 2.—The Voice After Laryngofissure—Continued

Patient	Sex	Age	Date of Operation	Have You Developed a Useful Voice?	Is Your Voice Good? Fair? Poor?	What Was Your Occupation?	Were You Able to Resume the Same Work?	Do You Talk on the Telephone?	Do You Address Groups?	Did You Sing?	Are You Able to Sing Since Operation?	Comment
35. W. C. V.	M	54	4/12/34	Yes	Fair	Buyer and seller of merchandise	Yes, but weaker voice has been a handicap	Yes	No	Yes	Yes, in a low tone; not much volume	
36. H. R. W.	M	71	6/ 7/32	No	Poor	Farmer	Handicapped	No	No	No	No	"My voice has improved very little since I left the hospital in July 1932. It is an effort and strain to talk so as to be understood."
37. H. L.	M	57	7/12/32	Yes	Fair	Lumberman	Yes	Yes	Small groups	No	No	
38. H. McD.	M	61	8/23/35	Yes	Good	Farmer	No, because of other ailments	Yes	Yes	A little	No	"My voice is much better than I thought it would be, and I can talk so that I can be heard by a group of people or a person in the same room. When I am speaking, though, my voice lowers at the end of every sentence. I am much pleased with the operation and grateful."
39. H. S. McK.	M	70	10/15/36	Yes	Fair	Office worker	Yes	Yes	Small groups	No	No	"About the first of the year (1939) I noticed it becoming increasingly difficult to talk in a normal way and developed an inclination to whisper; I am still doing this."
40. W. M.	M	47	7/ 3/34	No	Fair	Pharmacist	No	Yes	No	No	No	"Those who know and are used to my voice understand me over the telephone but the greater percentage do not. I was not able to resume the same work so far as the use of the voice is required."
41. L. P.	F	46	3/ 6/37	Yes	Fair	Saleswoman	No	Yes	No	Yes	No	"A recent examination by a local specialist showed no change in condition, and my voice is much stronger. After I have talked a great deal my throat feels tired; then whispering takes place."
42. A. A. S.	M	55	10/ 2/35	Yes	Fair	Industrialist	Yes	Yes	No	No	No	"I am able to talk on the telephone, but with difficulty."
43. H. B. T.	M	54	2/17/31	Yes	Good	Sales manager	Yes	Yes	Yes	No	No	"Some of my old friends maintain that my voice continues to become more normal, and I find, especially in making sales over the long distance telephone, that I have practically no trouble in making myself heard. I feel most grateful to you for what you have done to make it possible for me to carry on, hold the same position and enjoy good health."
44. H. E. W.	M	45	4/12/33	No	Fair	Head of production department	No	A little	No	No	No	
45. H. Y.	M	57	8/ 9/34	Yes	Fair	Window cleaner	Yes	Yes	Occasionally	No	No	"I find a definite improvement in my voice since my operation."

44. H. E. W.	M	45	4/12/33	No	Fair	Head of production department	No	A little	No	No	No	
45. H. Y.	M	57	8/ 9/34	Yes	Fair	Window cleaner	Yes	Yes	Occasionally	No	No	"I find a definite improvement in my voice since my operation."
46. C. M.	M	60	9/28/38	Yes	Fair	Engraver	Yes	When necessary, but only fairly well	No	Not for about a year before	No	"My voice seems increasing in strength. Before operation I could speak only slightly above a whisper."
47. R. L. W.	M	50	13/11/31	Yes	Good	Manager of telegraph office	Yes	Yes	No	Yes	No; hesitate to put voice to test	"I feel that I have successfully used my voice in my work, meeting my public and using the telephone as my work calls for. My voice is improving all the time. I had a singing voice with a great deal of volume but not cultivated. I used it only for the pleasure of my family and friends. I have not been able to sing since the operation. Perhaps I could develop some sort of singing voice if I were not afraid of straining."
48. G .N. L.	M	47	9/25/37	Yes	Fair	At time of operation, Secretary of State for Foreign Affairs of ——; now, lawyer	Yes	Yes	Yes	No	No	"I was called on a few months after operation to make frequent public speeches to ———— Congress and was able to do so without any difficulty with the use of an amplifier. In the month of April 1938 I was able to speak in public without an amplifier but it was naturally a strain on my voice."
49. T. G.	M	58	10/29/38	Yes	Good	Salesman	Yes, to a large extent	Yes	No	No	No	"While my voice might be considered good it is, of course, not strong, and it is an effort to talk if there is any other noise or conversation going on. I cannot talk where machinery is in operation, but you advised me not to do that or to strain my voice; in a quiet room I have no difficulty at all. I can even talk to a person in the next room if there is no other noise, and I really feel that my voice is gradually getting stronger."
50. H. W.	M	61	4/22/37	Yes	Poor	Executive of shoe manufacturing company	Not to same extent	At times, but with more or less difficulty in being heard, for which reason I avoid it except when necessary	No	No	No	"I try to avoid meetings or groups that are noisy, as I find that trying to talk over noises requires some straining, resulting in irritation and more or less coughing. This condition is also caused if I talk too long at one time, even though it may not be necessary to raise my voice."
51. E. E. H.	M	54	3/11/38	Yes	Fair	Physician	Yes	Yes	With great difficulty; I am unable to speak loud	No	No	"In ordinary conversation my voice is husky and unmusical, but I get along satisfactorily. I cannot talk loudly enough to be heard over noises like a room full of people in conversation, nor can I talk to one across a room with comfort."

TABLE 3.—*The Voice After Laryngectomy*

Patient	Sex	Age	Date of Operation	Have You Learned to Speak Without the Larynx?	Is Your Voice Good? Fair? Poor?	What Was Your Occupation?	Were You Able to Resume Your Work?	Have You Tried an Artificial Larynx?	Was it Satisfactory?	Comment
1. A. B.	M	54	3/14/35	Yes	Fair	Dealer in fruits and vegetables	Yes	No	...	
2. W. I. B.	M	62	2/11/36	Yes	Good	Salesman	No	No	...	"I was unable to resume work because no position is available because of my age. I could not talk fast anyway. I can carry on ordinary conversation comfortably."
3. L. H.	M	66	4/ 6/34	No	Poor	Merchant and cotton ginner	Yes	Yes	Yes	"I firmly believe that if I had not been so impatient and had let you work on my throat another day or two I should have been able to use my voice as I was doing before. I am as healthy as any one can be."
4. M. K.	M	47	1/24/33	Yes	Good	Grocer	Yes	No	...	
5. E. L. K.	M	54	5/18/36	Yes	Fair	Carpenter	Yes	No	...	
6. F. W. K.	M	81	10/29/34	Yes	Good	Captain, U. S. N., retired	Retired	Yes	Fairly so	"I was taught to use the 'pseudovoice' by Dr. Morrison of New York. My voice varies. Prolonged talking is an effort and makes me cough."
7. J. H. L.	M	49	6/22/34	Yes	Good	Physician	Yes	No	...	"I am extremely grateful, as the operation saved my life and returned me to useful work."
8. M. L.	M	56	9/ 8/30	Yes	Good	Laborer	No, unemployed	No	...	"I should be a lot better if I could work. I feel well, if I only had a job to occupy my time."
9. P. K. L.	M	66	11/ 2/37	No	Fair	Steam and air fitter	Pensioned	No	...	"I weigh more now than ever. I have never tried to talk too hard because it is too hard on the stomach of an old man."
10. C. M.	M	66	6/ 1/34	No	Good	Molder	Worked 3 years	Yes	Yes and no	
11. L. N.	F	44	8/19/30	Yes	Good	Housewife	Yes	No	...	
12. Q. R.	M	65	10/ 1/36	No	Fair	Wood carver	Retired	No	...	
13. H. S.	M	65	1/15/37	Yes	Fair	Railroad engineer	No	No	...	"I use a whispered voice."
14. I. W.	M	58	6/10/38	Yes	Poor	Suitcase maker	Yes	No	...	"I imagine I could speak much better if I were able to swallow without its hurting me. It does not hurt to swallow food, only air."
15. F. Z.	M	67	12/ 1/31	Yes	Good	Magistrate	Yes	Yes	No	
16. E. D. B.	M	67	9/24/30	No, whisper	Good	Worker in steel business	Retired	Yes	Very	"I tried the Bell Telephone larynx without success and have used the Voco-Phone, produced by McKesson Appliance Co., Toledo, Ohio, since June 1931. Any additional information you may wish will gladly be supplied. I worked all the time from June 1931 to Dec. 31, 1937."

supplied. I worked all the time from June 1931 to Dec. 31, 1937."

17. J. H. C.	M	64	1/20/38	Very little	Poor	Printer	Was superintendent; now a compositor	No	...	
18. N. B.	M	57	11/23/31	Whisper	Poor	Tailor	No	No	...	
19. W. J. H.	M	53	12/28/37	Yes	Fair	Salesman	Yes	No	...	"My voice is exceptionally good."
20. M. M.	F	59	5/14/35	No	Poor	Housewife	Yes	No	...	"I can be understood by those used to conversing with me, but most of my conversation is carried on by writing. I have never used an artificial larynx. My general condition is good."
21. F. O.	M	70	1/22/37	No	Poor	Steel worker	Pensioned	No	...	
22. C. A. B.	M	51	7/14/31	Loud whisper	Fair	Hotel proprietor, 29 years	Only in a supervisory way	No	...	"As you remember, I told you before operation that I would come out of the operating room talking. I have not given up, and I am still trying, with the result that I have been able to carry on a conversation with persons who are around me often, so that they understand me very well."
23. G. C.	M	68	9/ 4/31	Yes	Good	President and owner	Yes	Yes, one time	Advised not to use it, since if I did I wouldn't learn to talk	"Dr. ——— looked at my throat this morning and said that it was all right and that I should tell you that my voice is good. I talk over the telephone and, while I think that I talk low, my voice seems to carry as persons upstairs can hear me from down stairs in the office (from 40 to 50 feet [12 to 15 meters])."
24. J. K.	M	59	12/20/37	Yes	Fair	Printer	Part time	No	...	
25. R. J. O.	M	49	7/19/32	Not very well	Fair	Waiter	No, working for city in small way	Yes	No	
26. J. P.	M	53	7/11/36	Yes	Fair	Unemployed	Unemployed	No	...	"I should speak much better than I do now if my voice did not escape through the opening left after the laryngectomy. I should be glad if you would help me in making my voice louder."
27. R. B. W.	F	56	11/ 4/30	No, only in a whisper	Poor	Housewife	Partially	Have Bell telephone artificial larynx but cannot use it owing to excessive discharge of mucus	No	"In the even climate of San Diego I am less subject to colds and pneumonia than in the East."
28. H. S.	M	47	2/ 1/39	Yes	Good	Milkman	Yes, but job taken by some one else	No	...	
19. S. H.	M	54	10/ 8/37	Yes	Good	Storekeeper	Yes	No	...	
30. T. McF.	M	54	1/24/39	Yes	Fair	Painter	Unemployed	No	...	

explains that it will be necessary for him to learn to collect air in his hypopharynx, esophagus and stomach to replace the air column from the lungs and to produce tone by means of vicarious cords formed in the hypopharynx or pharynx. He will probably be skeptical about this, but one can assure him that experience has shown how to help patients get the knack of this sort of speech and that his course of voice lessons will be begun as soon after the operation as healing permits. I believe that the material presented in the tables published herewith will afford considerable help in reassuring patients about to have laryngectomy done. Schall [8] has published two excellent papers on the subject. He stated, "The laryngectomized patient is not a social outcast, nor in most cases is he an economic burden." He has found that such patients constitute "a most optimistic, cheerful group, neither objects of curiosity nor, in most cases, recipients of charity."

These views regarding the social and economic rehabilitation of the laryngectomized patient are fully supported by the replies to the questionnaire which was sent out in this study. At the present time I have had replies from 30 patients operated on during the ten years, and of these 18 replied "Yes" to the question "Have you learned to speak without your larynx?" Five stated that they could whisper; only 6 said flatly "No." One of the most interesting things noted in the replies to the questionnaires was the fact that in reply to the question "Is your voice good, fair or poor?" 50 per cent of the laryngectomized patients replied "good," while only a little over 10 per cent of the patients submitted to laryngofissure replied "good," most of them saying "fair." The patients who had laryngectomy, classified as to occupation, were: salesmen, merchants, carpenters, army officers, physicians, laborers, steam fitters, molder, housewives, locomotive engineer, wood carver, magistrate, executive, printer, tailor, hotel proprietor, waiter, gate tender and milkman. Seventeen of these patients have been able to resume the same work, wholly or partially. Among the questions asked the laryngectomized patients was whether or not they had tried to use an artificial larynx and whether it had proved satisfactory. Only 8 had tried it, and only 2 found it satisfactory—1 other said "fairly so" and still another "yes and no."

Morrison [9] deserves a great deal of credit for the work he has done in advocating a definite course of vocal instruction for laryngectomized patients. Some do not require lessons, but others make little progress

8. Schall, L. A.: (*a*) Psychology of Laryngectomized Patients, Arch. Otolaryng. **28**:581 (Oct.) 1938; (*b*) Laryngectomy, Pennsylvania M. J. **41**:261 (Jan.) 1938.

9. Morrison, W. W.: The Production of Voice and Speech Following Total Laryngectomy, Arch. Otolaryng. **14**:413 (Oct.) 1931.

or develop hopelessly bad habits if they are not systematically taught. I first became interested in the subject when a patient from the clinic with which I am associated, who had considerable difficulty in learning to talk even after he had thoroughly grasped the fundamental principles of buccoesophageal speech, consulted Dr. Morrison, undertook a course of lessons with him and at once began to improve. Since then it has been the practice to have the patient begin voice lessons in each case as soon as the condition of the wound permits, and Dr. N. M. Levin, a member of the staff of the clinic, who has been particularly entrusted with this work, has been obtaining excellent results. I plan now to communicate with all those who have stated in their reply to the questionnaire that they have not been able to develop a satisfactory voice, requesting that they come in for instruction.

It is planned also to examine the entire series of patients with a view to determining what anatomic factors, if any, influence the voice. I have seen in some cases a "pseudoglottis," resembling the true glottis to a remarkable extent, while in other patients, having an equally good voice, it is difficult to ascertain what the vibrating structures are. There has been some question as to the advisability of leaving the epiglottis, with the idea that it might aid in the development of a voice. Personally I think this an unwise practice, because of the danger of incompleteness of removal of the tumor. As a matter of fact, I doubt the essential value of the epiglottis as a vibrator, because I have observed equally good voices in patients from whom the epiglottis had been removed.

With regard to the artificial larynx, I believe it is a mistake to have the patient try it before he has had a chance to develop a buccoesophageal voice. I believe its use should be reserved for those who are not able, after a fair trial, to develop this more natural "pseudovoice."

CONCLUSION

1. After direct laryngoscopic operations for the removal of benign tumors, the voice should, except in special cases, be restored to normal clarity.

2. Nodules of the vocal cords should be carefully removed by direct laryngoscopic procedure (or by indirect laryngoscopic procedure if preferred); however, in cases of professional singers it is perhaps best to try first a period of vocal rest and special exercises.

3. While voice is of secondary importance in procedures undertaken for the eradication of a malignant tumor, the reply to a questionnaire sent out to a series of patients who had had laryngofissure or laryngectomy for cancer of the larynx showed that the majority had been able to develop useful voices and to resume their original occupations.

4. The majority of the patients who had developed a voice after laryngectomy considered it "good," while the majority of patients who had had laryngofissure considered their voices only "fair."

5. While many patients acquire spontaneously the knack of buccoesophageal speech, it is best to give laryngectomized patients a systematic course of voice lessons, as advocated by Morrison, just as soon as the wound is healed.

6. The artificial larynx should not be tried until after the patient has made some effort to develop a voice without it.

DISCUSSION ON PAPERS IN SYMPOSIUM ON VOCAL DEFECTS

DR. WALTER H. THEOBALD, Chicago: I am wondering, in connection with the effect of faradic current applied to the neck, if Dr. Mithoefer knows that in an experiment at the Research Hospital by Dr. Fischmann and myself in no instance, in dogs or in man, were we able to record any change in the vocal cord whatsoever when faradic stimulation was given on the outside to any of the nuchal muscles.

I wonder if the effect is not, after all, psychic. It is known that when a person chins himself, pulling himself up by a handle bar, and uses the voice, it is promptly elevated.

I had hoped that Dr. Mithoefer would touch on the subject of a posterior chink in the cord that is lax and bowed. I should like to report to him that I have observed 6 cases now in which a posterior chink was present and weakness in the voice resulted from it and the escaping air. I found the 6 patients to have a low metabolic rate, varying from —16 to —26 per cent, and in each case by merely treating the patient with thyroid the posterior chink was closed.

DR. AUSTIN A. HAYDEN, Chicago: The large class of persons, it seems to me, that need vocal training or, at least, vocal improvement are generally not handled by the specialists in vocal improvement. They are the persons that do not hear well and that have lost some of the normality of their voices because they cannot hear themselves talk with perception of all the tones and therefore cannot control their voices satisfactorily to themselves and to the persons that hear them.

This is, of course, an ever present problem with any one who has much to do with hard of hearing persons.

DR. BURT R. SHURLY, Detroit: The work in the public school system in Detroit has been developed in a marvelous way. One of the first of the departments teaching tones to those with defects in speech was established, with the school for the deaf and the department of speech defect working harmoniously in cooperation and in consultation and then carrying on in the school system an extensive test for loss of hearing among the school children. The 12,500 children examined showed a 4.8 per cent loss of hearing.

Now, if such patients are to be reached it must certainly be in the early years of life. I feel that a widespread general examination for defects of hearing through the great school systems of the country would bring to the otologist many patients with such defects in the earlier years of life, during which there is some hope of doing something for them. The difficulties that arise after the troubles have continued for some time are well known.

DR. LOUIS H. CLERF, Philadelphia: I should like to ask Dr. Mithoefer if this faradic current therapy would be useful for disorders due to the excessive use of the larynx. Does he use the faradic current in the treatment of such disorders?

DR. HAROLD I. LILLIE, Rochester, Minn.: In many cases the vocal fatigue Dr. Mithoefer has talked about, as he has suggested, is from the wrong use of

the voice. There are certain persons who need to do considerable public speaking and try to force their voices beyond their natural capacity, and they get no relaxation. There is tension. If they would lower their voices, they would get complete relaxation, and their voices would carry miles.

Dr. James S. Greene, New York: In New York there is a League for the Hard of Hearing. I thought it would be a good idea to let the league take care of the persons who do not hear well. Therefore, Dr. Hayden will have to talk to Dr. Voorhees about the hard of hearing persons. Dr. Theobald wanted to know about treatment. It is rather difficult to tell what my colleagues and I do, but I have invited Dr. Theobald to come to New York to our clinic. I extend this invitation to any member here. We have no secrets. We are only too glad to show and tell what we do.

Dr. Irving Wilson Voorhees, New York: Mucous nodules can be cured by rest and by vocal exercise, but about fibrous nodules I am doubtful, although occasionally I find a singing teacher who says he can cure any patient with vocal nodules through exercises and once in a while a physician believes that that is true. My own feeling is that it is about as difficult to sing a nodule off a vocal cord as it is to walk a corn off a toe.

Dr. William Mithoefer, Cincinnati: I can't conceive of a psychic element in the treatment of phonasthenia with electrophonic compensation, because physiologically it is known that the note rises when the application is made. The posterior chink in the vocal cord of a hypothyroid patient probably is there because of mild infiltration. I am a great believer in giving a systemic treatment, if necessary, to singers, and if they do not improve after several treatments, I do not depend on the basal metabolic rate but depend on the afternoon temperature, between 4 and 6. If it is subnormal, especially if it is recorded for a week, I always advise thyroid medication, beginning with a small dose and increasing slowly until the afternoon temperature is normal or the pulse rate rises to 95.

Dr. James S. Greene, New York: It seems to me that Dr. Mithoefer does not fully realize, at least the way I see it, the interrelation between the psychic and the somatic, in other words, the "physiologizing" of the emotions.

The mere fact that the physician applies his little instrument is enough stimulus to change the patient psychically. I have seen that again and again. Dr. Voorhees will bear me out that on Friday or Saturday he talked to a patient who came to my clinic from Minneapolis. The patient had great difficulty with his speech, and he had had colitis for five years. He came to the clinic, and the only thing I asked him to do was to stop all treatment for his colitis while he was taking the speech treatment. Saturday he went home. He is speaking beautifully, and his colitis is absolutely cured, which indicates the "physiologizing" of the emotions.

The physician deals with temperamental persons, subject to elation and depression, so much so that I have taken it on myself in recent years not to call the condition phonasthenia any more. I have changed the name. I call it psychophonasthenia. The most important phase in such cases is the psychic.

DEFECTIVE SPEECH IN RELATION TO DEFECTIVE HEARING

MAX A. GOLDSTEIN, M.D.
ST. LOUIS

Perfect speech is dependent on perfect hearing, correct voice production, understanding of pitch, volume and rhythm control and careful articulation.

It is a reasonable and logical observation that defective speech is often the result of imperfect hearing. Speech in its essence and performance is but an imitation of the sounds of the human voice as heard.

Defective hearing may be the main factor in many cases of speech defects, notwithstanding good voice and careful articulation. A lack of physical appreciation of pitch, volume and rhythm, due to defective hearing, may counteract all favorable elements in the production of perfect speech.

Speech is the most constant and universal instrument of exchange in the social contacts of man with man; it is the one fundamental act that distinguishes the human being from brute creation; it is a vital element of social intercourse; it is the most useful and frequent form of expression of thought; it is the principal factor in advancement and success; it is the keystone of emancipation from many handicaps.

There are so many details to be considered in the discussion of speech and its defects that only a few features may be touched on in this short survey.

Voice is sound originated in the larynx and may be produced by any animal possessing that organ.

Speech is voice modified in the cavity of the mouth by changes in the size and shape of this cavity, controlled by the mechanism of articulation, comprising the tongue, teeth, lips, velum and mandible.

As speech is dependent on normal hearing for its perfect production, the model of speech must be perfect. If the model is imperfect and the hearing imperfect, the resultant speech will be imperfect.

There is an interesting story or legend ascribed to the Chinese: Some centuries ago a mandarin ordered a normal-hearing young child, before speech had been established, to be placed under the supervision of mute attendants on a deserted island. According to the story, this

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child grew up and throughout adolescence, notwithstanding his normal hearing, was unable to speak.

Among disorders of speech of childhood, the outstanding and most frequently to be considered etiologically associated with defective hearing is dyslalia, also known as oral inactivity, or, more commonly, as "baby talk." Dyslalia is defined as "Impairment of utterance with functional abnormality of the external speech organs" and includes a great variety of articulatory defects.

In the examination of many patients the origin of such defects can be traced to imperfectly heard speech patterns or imitation of fond mothers and relatives who coo and distort their own speech for the child's and their own amusement, little realizing that they are setting an example and offering every possible opportunity for the development of this prevalent speech defect. In addition to this, it may be observed in passing that the parents of such a child frequently consult a pediatrician about the prospects of correcting this defect, only to be told by him, "Don't worry, Mrs. Smith, your child will outgrow it."

The second chapter in the program of dyslalia (baby talk) is frequently ushered in by the appearance of acute stammering.

Speech correctionists refer constantly to dyslalia as a frequent forerunner of stammering.

Another but less common etiologic factor in speech defects is word deafness, a central disturbance of the auditory center in the cortex, in which sounds are heard but convey no meaning to the child.

In describing the speech centers the Wernicke center is referred to as the speech-hearing center and the Broca center as the speech-producing center. In word deafness, therefore, the Wernicke center may be delayed in development, or its relation to the Broca center by association fibers may be impaired.

In young children both dyslalia and word deafness are thoroughly amenable to correction in the hands of expert speech correctionists.

In the later nomenclature, the congenitally deaf child has been constantly referred to as the "deaf-mute." This has been a general and all-inclusive term that has been applied throughout the ages to persons with almost every form of deafness, congenital or acquired, in which the social and economic helplessness of the affected person has been complicated by inability to speak. Even in the present era of better understanding and oral training for the deaf, the term "deaf-mutism" is still applied more or less indiscriminately.

Congenital deafness of the profound type (with no residuum of hearing) and congenital mutism rarely occur simultaneously. It has been my experience that congenital mutism per se is extremely rare.

With the advent of methods of oral instruction by which the young congenitally deaf subject can be taught the mechanics of rhythm, pitch,

accent, volume and quality of speech by the application of sight and touch as a substitute for the lost sense of hearing, the claim is now justifiable that every congenitally deaf child has the potentiality of speech. All that is required is the opportunity for such instruction under the guidance of specially and properly qualified teachers.

Another defect in speech which accounts for a large class of physically handicapped children is stammering and stuttering. Psychology and pedagogy have emphasized the significance of heredity and environment as causal factors in these speech defects. The clinical and otolaryngologic examinations that are a routine in speech clinics devoted to the correction of stammering in children demonstrate that sizable numbers of such children have substantial mechanical defects in the nasal or the oral cavity. Even in this rabid age of indiscriminate tonsillectomy one finds surprisingly large numbers of patients in such clinics with enormously hypertrophied faucial tonsils and adenoids, in addition to those with nasal obstruction due to marked septal deflection, turbinal occlusion or chronic sinusitis.

It is unnecessary to discuss before the American Laryngological Association the physical obstructions to which such pathologic conditions may lead as far as hearing is concerned. It is the practice to include in a complete clinical inspection an individual audiogram and both Weber and Rinne tests with the tuning forks. The results show corroborative evidence of defective hearing in varying degrees, often incapacitating the pupil for acceptable scholarship in the normal classroom.

Even when the tonsils and adenoids have been expertly removed there may be recurrent lymphoid tissue in both the posterior and the lateral part of the nasopharynx, interfering with the normal functions of the eustachian tube and sometimes even affecting the labyrinth by low grade toxic absorption.

The care of the child with such a condition may be classified distinctly as preventive medicine, and in connection with this thought I offer the observation that many of the group surveys of school children have been substantial factors in establishing deafness as a cause of speech defects. The survey, however, cannot be considered complete without the follow-up.

To cite one of many cases in question: In the course of an individual clinical examination a child is found who reveals hypertrophied or diseased tonsils, recurrent adenoids or nasal obstruction of a character that leads the otolaryngologist to conclude that the hearing or even the general health of the child warrants surgical intervention. Recommendation is made to the parent through the school physician or school nurse that the child should be taken to a laryngologist or visit a laryngologic clinic for such operative service. The unfortunate hitch

in this procedure is the objection of the parent, either through ignorance or through aversion to operation. It has been frequently reported by the school nurse or school physician that the parent is adamant to these suggestions. There is no law of school, municipality or state that can compel a parent to accept such advice, and the parent is thus placed in the unenviable position of acting as an obstructive influence to the health and future of his own child.

Much educational stimulus may be given by the laryngologist concerned with the welfare of the school child by appearing before mothers' groups and parent-teacher associations and explaining the significance and importance of these problems.

Another practical observation to be cited in connection with defective speech in its relation to defective hearing is the case of the deafened adult whose speech quality, articulation, pitch and volume suffer materially when his hearing becomes so defective that he is unable to hear his own voice distinctly and is therefore unable to make the necessary correction of these various speech deficiencies.

Surveys that have been instituted in school systems throughout the nation, stimulated and developed by otolaryngologists, have had many important and far-reaching results and have awakened the larger communities to the significant fact that over 3,000,000 of the 50,000,000 pupils enrolled in the public school systems of the United States of America have imperfect hearing and 4,000,000 demonstrate some defect in speech. These defects range from slight to serious handicaps. When as many as 14 per cent of the future citizens of a vast nation show defects in speech or hearing, a large percentage of which can be definitely and permanently corrected, it becomes not only a question in which otolaryngologists and physicians are concerned but also one in the careful study of which every vital agency of the nation must be interested.

It is a frequent observation that Americans have inherited the English language as one of their most splendid assets from the English people and an equally frequent and justifiable criticism that they have distorted and often destroyed this asset by the introduction of slang, vernacular and slovenly speech.

If more care and attention were given to the development of oral English for the benefit of the rising generation, I venture the thought that some of the less potent cultural elements that have been injected into the school curriculum could well be dispensed with.

I think enough has been said in this brief paper to justify the conclusion that the problem here considered is vital and that it is distinctly the province of the laryngologist to clarify the attitude of the parent, to emphasize proper surgical treatment in the individual case and to create a better understanding of the principles of preventive medicine.

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DISCUSSION

DR. GEORGE B. WOOD, Philadelphia: I have heard of two children of early age who were deserted by their parents and allowed to run wild. They developed a language sufficient for communication with each other.

DR. GORDON BERRY, Worcester, Mass.: There is one brief thought that I might contribute in connection with Dr. Goldstein's statement that some children who needed surgical procedure could not have it done because the parents would not permit. The legal phases come in there. If the help of a probate judge is secured and the condition is one of real emergency, the purpose can be accomplished.

DR. BURT R. SHURLY, Detroit: The relation of speech to hearing suggests one of the great works that are being done in Detroit. The school system established one of the first speech correction departments in the United States. It has been remarkably successful and is becoming more and more fully appreciated by parents.

A resolution was introduced at a meeting of the American Medical Association asking for the encouragement and support of the medical profession in the endeavor to test children for defective hearing. In the last 12,500 tests performed by representatives of the board of education and the board of health of the city of Detroit 4.8 per cent of the subjects were found to have defective hearing.

As the relation of the nose and throat to many defects of speech and hearing is so important from the standpoint of laryngologists I would make a plea for a nationwide testing of school children. Dr. Goldstein has brought out that three million children are handicapped by such defects. The work is necessary if these physically handicapped children are to be educated. It is a great expense to a board of education to look after children who cannot see properly, are not properly fitted with glasses, cannot hear or have tonsils and adenoids that should be removed.

I believe that the rise and fall of popular opinion shows a need for a little reeducation, because the pendulum swings, as interest in many other ideas swings in America. Those who are interested in the administration of boards of education feel keenly that the backing of the medical profession is needed to give parents a better idea of the importance of the physical handicaps of children.

It is necessary only to speak of some of the education against operation to make me feel that all laryngologists should get together again and develop the care of the handicapped child that their specialty is concerned with.

Recent reports by Dr. Crowe and his associates show that definite loss of acuity of hearing for the higher frequencies (i. e., 4096 and 8192 double vibrations) often occurs in young children as the first indication of a conductive impairment of hearing due to obstructive lymphoid hyperplasia in and about the eustachian tubes. This is contrary to traditional teaching. In view of these findings it seems important to consider a revision and refinement of the technic for the early detection of deficiencies in hearing in children.

In many communities the 4-A or 4-B Western Electric phonograph audiometer or its equivalent has been extensively used to disclose deficiencies in hearing in school children by a screening process covering all pupils old enough to write dictated numbers. In two states, New York and Pennsylvania, its use is mandatory. It has recently been found by G. Oscar Russell, Ph.D., of the University of Ohio, at Columbus, Ohio, and by others that this test does not disclose deficiencies of hearing for the higher tones.

I would suggest, therefore, that the younger groups of school children be individually tested with a simple, accurate, quickly operated, pure tone, or pitch range, audiometer, such as has lately been made available at a low price. A preliminary trial by this method in the public schools of Minneapolis has disclosed a surprising number of children in the lower grades, not revealed by the usual tests, who have losses of hearing for the higher frequencies and who should have otologic scrutiny.

Yesterday in its business meeting the American Otological Society, following the example of the Section on Laryngology, Otology and Rhinology of the American Medical Association and other organizations, adopted a resolution to encourage and promote the granting of funds by foundations and appropriate agencies to make possible surveys, especially in rural areas, for the discovery of children who have or are threatened with defects in vision, hearing or speech and, in the case of the underprivileged and indigent, to provide corrective treatment through the cooperation and leadership of qualified physicians who are members of national, state and local medical societies.

I respectfully urge that because of the laryngologist's broad interest in the interrelation of speech and hearing, so ably brought out by Dr. Goldstein, a similar resolution should be adopted at the business meeting of the American Laryngological Association.

DR. WILLIAM E. GROVE, Milwaukee: Mr. Chairman, Dr. Goldstein spoke of the prevalence of enormously enlarged tonsils and adenoids in connection with the problem he discussed. Now that tonsillectomy and adenoidectomy in most parts of the country are performed not by otologists, rhinologists and laryngologists but by general practitioners and surgeons, poor operative work is being done. The tonsils are still pulled out with some success, although one sees all sorts of injuries to the pillars, the soft palate and other structures and many times the soft palate and the pilars are removed and the tonsils are left in.

To my mind, the most poorly performed operation in the field of laryngology is adenoidectomy. I am sorry to say that my statement includes some of the operations performed by laryngologists and certainly all of those performed by general practitioners who attempt to take the enlarged adenoid tissue out. I think a great deal of education in the proper performance of adenoidectomy should emanate from otolaryngologists.

DR. WILLIAM B. CHAMBERLIN, Cleveland: I merely wish to emphasize the thought that Dr. Shurly has expressed in regard to the universal examination of school children.

DR. THOMAS E. CARMODY, Denver: Whenever I operate for cleft palate I invite my confreres and pediatric friends to view the adenoid area. If they see where the adenoids are located, better work may be done.

DR. MAX A. GOLDSTEIN, St. Louis: I think Dr. Shurly sounded the keynote for the future and indicated the responsibility of the laryngologist in the rather important phase of development and improvement of the physical condition of the school child which has been discussed by referring to and emphasizing the importance of getting a change in the trend of public opinion. After all, it is public opinion that sways almost every large movement in a big nation. Not only closer cooperation by laryngologists is required but better understanding of the problem of the laity.

Incidentally, I might disclose a little secret, that the reason or one of the reasons (and let me say this without much ego) that the Detroit public school

system has shown such a splendid front in dealing with this problem is that until about three years ago every teacher of the day school for the deaf in Detroit had passed through the hands of my colleagues and me in the Teachers' Training College of Central Institute and was qualified as a teacher of the deaf and a teacher of speech correction. The results depend largely on the qualification of the teachers.

The suggestion made by Dr. Berry is worthy of further development. I am really seriously concerned as to how to go about getting better cooperation from parents. If it can be done by legal procedure or some other persuasive act, as the temporary appointment of a guardian to see that the suggestions of the laryngologists are carried out in cases in which it is vital and in which further defect in hearing and in speech can be prevented, it will be excellent.

RELATION OF ALLERGY AND TONSILLECTOMY IN CHILDREN

INCIDENCE OF RESPIRATORY ALLERGY IN CASES OF ROUTINE TONSILLECTOMY

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The otolaryngologist, the pediatrician and the general practitioner are frequently confronted with the problem of diagnosis and treatment in cases of recurrent colds, sinusitis, bronchitis and recurrent pneumonia in children. These conditions are generally considered as infectious, and the treatment usually consists of the administration of cold "shots," drainage of sinuses or the removal of the tonsils and adenoids. That these symptoms and signs may be of allergic instead of infectious origin has been emphasized by Piness and Miller, Cohen and Rudolph and Hansel. The allergic nature of these complaints is often not recognized until frank bronchial asthma develops.

Piness and Miller [1] pointed out that allergy alone may produce chronic nasal obstruction without sneezing, unassociated with enlarged adenoids, and may cause frequent attacks of rhinorrhea and nasal obstruction in no way related to infection, although often designated as colds or sinusitis. Allergy may also cause persistent sore throat unrelated to tonsillitis and stuffy ears unrelated to pharyngitis. A chronic or intermittent cough without infection of the bronchi and with absence of dyspnea and wheezing, or recurrent bronchopneumonia with fever and leukocytosis and without wheezing and the presence of pneumococci may also result from allergy. In other words, Piness and Miller emphasized that allergy may simulate many of the symptoms and signs of acute or chronic respiratory infection.

From the Department of Otolaryngology, Washington University School of Medicine.

1. Piness, G., and Miller, H.: Allergy of the Upper Respiratory Tract in Infancy and Childhood, J. A. M. A. **113**:734 (Aug. 26) 1939.

It has been pointed out by Cohen and Rudolph [2] and by Hansel [3] that the commonly occurring conditions of the respiratory tract in children should be considered as one of the following types: (1) allergic, (2) infectious or (3) combined allergic and infectious. The symptoms in all three types are often similar; hence differential diagnosis depends on careful clinical and laboratory investigations. In table 1 are listed

TABLE 1.[2]—*Differential Diagnosis of Allergic and Infectious Conditions of the Upper Respiratory Tract in Children*

Allergic	Infectious
History	
1. Attacks usually recurrent	1. Attacks usually single
2. Often mild symptoms between attacks	2. Usually clears up completely
3. Definite relation to heredity	3. No relation to heredity
4. Not contagious	4. Contagious
5. Not related to exposure to another case	5. Definite relation to exposure to another case
6. Constitutional symptoms slight	6. Constitutional symptoms more marked
7. Foods and inhaled substances often traced as causes	7. No relation to foods or inhaled substances as cause
8. Itching common	8. No itching
9. Wheezing common	9. No wheezing
10. Other allergic conditions present or in past history	10. Usually no other allergic condition present or in past history
Examination	
1. Visible mucous membranes pale, glistening, edematous	1. Visible mucous membranes, hyperemic, red
2. Thin, watery, mucoid nasal discharge; mucous sputum	2. Mucopurulent or purulent nasal discharge or sputum
3. Smear shows eosinophils in large numbers	3. Smear shows polymorphonuclear neutrophils as predominant cell, eosinophils few or absent
4. Other signs of allergy often present	4. No other signs of allergy
5. Sinus involvement of edematous type	5. Sinus involvement of purulent type
6. Wheezing breath sounds	6. No wheezing breath sounds
7. Roentgenogram shows bronchial markings increased	7. Bronchial markings not increased in roentgenogram
8. Allergic skin reactions usually positive	8. Allergic skin reactions usually negative
Treatment	
1. Epinephrine specific for asthmatic symptoms	1. No relief from epinephrine or ephedrine
2. Avoidance of specific allergens followed by relief	2. Avoidance of food or inhalant substances produces no change

the differential points of allergic and infectious conditions as reported by Cohen and Rudolph.

The combined allergic and infectious type is frequently difficult to diagnose at the first observation. Acute and chronic infections may obscure the allergic phase of the condition.

2. Cohen, M. B., and Rudolph, J. A.: Allergic and Infectious Conditions of the Upper Respiratory Tract in Children: Differential Diagnosis, J. A. M. A. **97**:980 (Oct. 3) 1931.

3. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses, St. Louis, C. V. Mosby Company, 1936.

The incidence of respiratory infections during the first five years of life was investigated by McLean.[4] A group of 156 children was studied. Fifty-one of the patients were subjected to removal of the tonsils and adenoids. It was found that this procedure had little or no influence on either the number or the severity of respiratory infections. The average incidence of colds in the entire group was 8.04, and the average period between colds was one hundred and eighty-one days.

An extensive investigation of the relation of the tonsils and adenoids to infection in children was conducted by Kaiser.[5] His observations were based on a control study of 4,400 children over a ten year period. It was concluded that the removal of the tonsils and adenoids favorably influenced the incidence of head colds. The incidence among tonsillectomized children was 22 per cent, as compared with 31 per cent for the controls. On the other hand, the incidence of bronchitis was somewhat increased among the children who had the tonsils and adenoids removed. It was also noted that pneumonia occurred more frequently in this group. The effect of the operation also had an unfavorable influence as far as sinusitis was concerned, for it was noted that first attacks of sinusitis occurred somewhat more commonly in these children.

In the evaluation of statistical studies on the incidence of respiratory infections in children, emphasis should be placed on the importance of excluding or taking into consideration those patients who have allergic respiratory symptoms which simulate the infectious type. Definite statistical error is evident if allergic patients are considered as having infection. Children with nasal allergy, allergic involvement of the sinuses, allergic bronchitis and allergic pneumonia will not be favorably influenced by tonsillectomy.

As patients are often seen for the first time when the symptoms are more pronounced, it is always a question whether the acute exacerbation is allergic or whether it is an acute, subacute or chronic infection. Repeated observations of the patient with especial reference to the cytologic character of the secretions may be necessary in all cases before an accurate diagnosis can be established. It must be definitely determined, therefore, whether the respiratory condition is allergic, infectious or allergic complicated by acute or chronic infection.

Unless a definite plan of investigation is followed in these cases errors in diagnosis are likely to occur.

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4. McLean, C. C.: The Incidence of Respiratory Infection During the First Five Years of Life, Arch. Pediat. **49**:279, 1932.

5. Kaiser, A. D.: The Relation of Tonsils and Adenoids to Infections in Children, Based on a Control Study of Forty-Four Hundred Children Over a Ten Year Period, Am. J. Dis. Child. **41**:568 (March) 1931.

In the management of respiratory infections in children, removal of the tonsils and adenoids is perhaps the procedure most commonly advised. It is most important, however, to separate the cases of respiratory allergy from the group of infections. Among the children observed routinely at the clinic of the Washington University School of Medicine for consideration of removal of the tonsils and adenoids, we have been impressed with the high incidence of respiratory allergy. In order to obtain some idea of the relative incidence of allergy in this group, we made a special analysis of 200 consecutive routine cases. The patients were observed during July, August and September 1938. Of the 200 patients 104 were boys and 96 girls. The ages varied from 3 to 16 years (table 2 and chart).

Of the 200 patients, 26 proved to have nasal allergy, an incidence of 13 per cent. Of the 26 patients with this condition, the local nasal symptoms were typical in only 13 instances. The complaints in all 26 cases are listed in table 3.

A positive family history of allergy was noted in 11, or 42 per cent, of the 26 cases, as follows: asthma, 6; hay fever, 3; asthma and hay fever, 1, and nasal allergy, 1.

A positive (past) history of other manifestations of allergy preceding the onset of nasal symptoms was obtained in 5 instances. Three patients had urticaria, and 2 had eczema.

In the local examination of the nose, it is noteworthy that only 14, or 54 per cent, of the patients with nasal allergy showed typical pallor or edema of the nasal mucosa. It is apparent, therefore, that diagnosis often cannot be established on the basis of the local changes.

In the 26 cases of nasal allergy the final diagnosis was based on the demonstration of eosinophils in the nasal secretions.

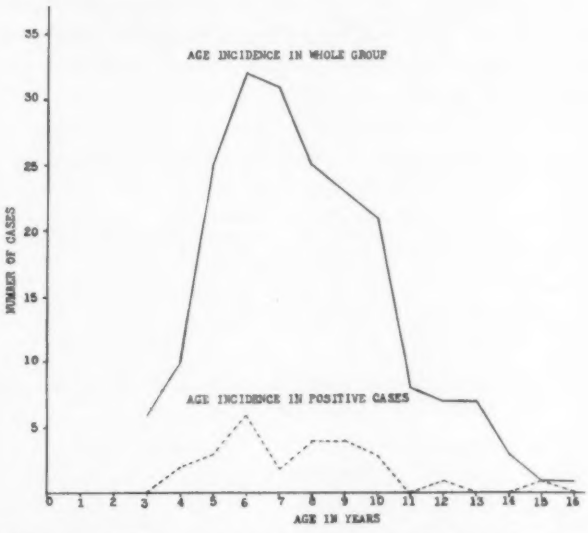
It is also noteworthy that 6 of the 26 patients had hay fever—3 due to grass and 3 to ragweed.

Of the group of 174 patients without nasal allergy, a positive family history of allergy was noted in 43, or 24.7 per cent. A positive past or present history of other forms of allergy was obtained in 18 instances, as follows: urticaria, 14 cases; eczema, 3, and eczema and urticaria, 1. In cases of other manifestations of allergy it is important to keep the patients under observation from time to time for the possible development of respiratory allergy. This is especially true of very young children, as respiratory allergy may be precipitated after an acute infectious disease, especially pertussis.

Of the 26 patients with nasal allergy 17, or 65.3 per cent, had already had an acute infectious disease (table 4).

TABLE 2.—*Age Incidence in 200 Cases of Respiratory Allergy*

Age, Yr.	Number in Entire Group	Number in Group with Nasal Allergy
3	6	0
4	10	2
5	25	3
6	32	6
7	31	2
8	25	4
9	23	4
10	21	3
11	8	0
12	7	1
13	7	0
14	3	0
15	1	1
16	1	0
Total	200	26



Age incidence in 200 cases of nasal allergy. In the chart, the number of cases in each age group from the group as a whole is indicated by a solid line; the number of cases of nasal allergy in each age group by a broken line.

In 1925 a survey of 840 allergic persons, mostly children, made by Piness and Miller [6] showed that the tonsils had been removed in 340, or 40 per cent. In 1938 a similar survey showed that of 2,230 allergic children 751, or 33 per cent, had had the tonsils removed. The combined reports of Cunningham,[7] Hansel,[8] Clarke and Rogers [8] and Waldbott,

6. Piness, G., and Miller, H.: Allergy: A Nonsurgical Disease of the Nose and Throat, J. A. M. A. **85**:339 (Aug. 1) 1925.

7. Cunningham, R. L.: Normal, Absent and Pathologic Tonsils in Young Women: A Comparison of Histories, Arch. Int. Med. **47**:513 (April) 1931.

8. Clarke, J. A., Jr., and Rogers, H. L.: A Statistical Study of Allergic (Vasomotor) Rhinitis, Arch. Otolaryng. **25**:124 (Feb.) 1937.

Ascher and Giese [9] showed that in a group of 2,186 allergic patients the average incidence of tonsillectomy was 38 per cent. Statistical studies by Cunningham [7] on 7,958 college women, between the ages of 15 and 19 years, revealed that the tonsils had been removed in 2,756, or 34.6 per cent.

Piness and Miller [1] further stated that it is a common belief that the signs and symptoms of allergy are due to infection. In a group of 273 children with bronchial asthma, they found that 119, or 43 per cent, had been advised that tonsillectomy would relieve the asthma. No proof was found to indicate that tonsillectomy relieved these patients. The fact that 751 children reported at the allergy clinic conducted by Piness and Miller after tonsillectomy had been performed is evidence that the procedure failed to give relief. It was further noted that in 116, or 17 per cent of 685 allergic children the first frank allergic symptoms developed after tonsillectomy, and many others complained that mild symptoms had been aggravated.

TABLE 3.—*Complaints and Symptoms in Cases of Nasal Allergy*

Nasal obstruction, discharge, sneezing, itching of nose, etc.	10
Headache	2
Sore throat and sneezing	1
Frequent infections of upper respiratory tract	1
Nasal obstruction and discharge	9
Sore throat	2
Swollen cervical glands	1
Total	26

TABLE 4.—*Occurrence of Acute Infectious Diseases in Twenty-Six Cases of Nasal Allergy*

Disease	No. of Cases
Measles	6
Scarlet fever	2
Pertussis	2
Parotitis	2
Chickenpox	1
Scarlet fever and pertussis	2
Measles and pertussis	2
Total	17

Our observations agree with those of Piness and Miller that tonsillectomy is often recommended for the relief of the symptoms of allergy because they have been mistaken for those of infection. In the absence of hay fever and asthma, nasal allergy and allergic bronchitis are frequently undiagnosed.

9. Waldbott, G. L.; Ascher, M. S., and Giese, F. W.: Results of Tonsillectomy in Allergic Patients, J. Michigan M. Soc. **35**:369, 1936.

Piness and Miller noted in the reexamination of 544 allergic children from whom the tonsils had been removed that 71, or 13 per cent, showed an excessive regrowth of lymphoid tissue in the tonsillar fossae.

According to a recent survey by Collins,[10] 1,235,000 tonsillectomies are performed each year in the United States. If one considers the incidence of allergy among patients having routine tonsillectomies as 13 per cent, about 160,000 allergic patients are subjected to this procedure in the United States per year.

Although tonsillectomy is indicated in certain patients who have nasal allergy, it is important to bear in mind that this procedure will not materially influence the allergic symptoms. The indications for tonsillectomy in children with nasal allergy should be the same as those in children without nasal allergy. In children with nasal allergy it is hardly possible to determine the incidence of infectious colds unless repeated clinical observations are made over a period of one to two years, during which time the cytologic character of the nasal and sinal secretions is correlated with the history and physical findings. Our observations on allergic children show that the highest incidence of colds occurs during the ages of 6, 7 and 8 years. It has been our experience that the majority of allergic children do not have more than the normal number of colds and that sinusitis is not a common complication.

By means of roentgenographic studies of the nasopharynx of children with nasal allergy, Cohen [11] has shown that edema of the adenoids is not uncommon. Subsidence of this edema was noted as a result of satisfactory management of the allergy. Enlargement of the adenoids in these cases, therefore, is not always an indication for surgical intervention.

The relative value of tonsillectomy in allergic children has been presented also in a recent report by Waldbott and his associates.[9] In a group of 1,112 patients with hay fever and asthma studied by them, tonsillectomy was performed in 228 before the onset of allergic symptoms and in 205 after allergic manifestations had developed. Of the latter group, definite relief occurred in 1.9 per cent, temporary relief in 1.9 per cent and aggravations of allergic symptoms in 11.6 per cent. In a control group of 60 patients subjected to tonsillectomy after they had been under observation the results were about the same. When tonsillectomy was performed for conditions other than allergy, it was successful in 35.2 per cent of the allergic group and in 36.4 per cent of the preallergic group, as compared with 72 per cent of the normal

10. Collins, S. D.: Frequency of Surgical Procedures Among Nine Thousand Families, Based on Nationwide Periodic Canvasses 1928-1931, Pub. Health Rep **53**:587, 1938.

11. Cohen, M. B.: Personal communication to the authors.

controls in whom improvement was recorded. Of 226 patients subjected to tonsillectomy before the onset of allergic symptoms, allergic manifestations developed in 14.4 per cent within three months, in 29.3 per cent within six months and in 48.6 per cent within two years after the operation. This is comparable with 25 per cent showing frankly allergic symptoms within three months, 47 per cent within six months and 63 per cent within two years. Of the patients whose tonsils were removed for the relief of nasal colds and bronchitis, tonsillectomy was of more benefit in the earlier age groups. The operation was less successful when performed during the pollen season. Although the results of tonsillectomies were disappointing, Waldbott and his co-workers expressed the belief that tonsillectomy should be performed in patients having frequent infections. In others with allergic catarrh of the upper part of the respiratory tract, which may or may not involve the tonsils, they concluded that tonsillar tissue is a definite asset to the system and should be preserved.

SUMMARY

1. In the management of frequent colds, sinusitis, bronchitis and recurrent pneumonia the possibility of allergy as an etiologic factor must always be considered.

2. Of 200 children considered as a routine for removal of the tonsils and adenoids because of these complaints, a definite diagnosis of nasal allergy was established in 26, or 13 per cent. It is significant that 6 of these patients had hay fever.

3. These cases of respiratory allergy may be overlooked unless an adequate history, physical examination and cytologic studies of the nasal secretion are carried out in each instance.

4. Removal of the tonsils and adenoids in allergic children should not be performed during hay fever seasons, and the indications for operation should be the same as in nonallergic children.

5. The tonsils and adenoids should not be removed with the idea of alleviating allergic symptoms.

ALTERATIONS IN NASAL FUNCTION DUE TO ANATOMIC VARIATIONS OF THE NARES

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The importance of the inspiratory and expiratory air currents in nasal function has been firmly established for many years. Goodale [1] and Lambert Lack [2] proved by their experiments that a stream of inspired air does not pursue a straight course from nostril to choana but passes in a wide curve which begins at the nostril, extends through the olfactory fissure and ends in the upper part of the choana. The current of air does not touch the inferior turbinate except for small eddies which curl (1) against the face of the sphenoid, (2) over the inferior turbinates and (3) into the sphenoid sinus.

At a much later date the experiments by Proetz,[3] using litmus paper and smoke trails, corroborated these observations. He also added a distinct inspiratory eddy of air in the nasofrontal angle.

Proetz's experiment with smoke brought out an important factor for clinical consideration. It was apparent from watching the smoke trails in the nose that the direction of the air current is determined by two factors. The first is the shape and position of the inlet (nostril) and the second the angle at which the inspired air impinges against the slope of the bridge of the nose; the choana, being larger than the nostril, does not modify the flow or direction of the current as it enters.

Further investigations by Proetz revealed other significant data. Thinness, shrinking or amputation of the middle turbinates does not materially alter either the inspiratory or the expiratory currents. But when obstacles are introduced either on the septal or the lateral wall, pronounced changes are observed, and the entire current of air is broken into numerous eddies.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

1. Goodale, cited by Thomson, St. Clair: Diseases of the Nose and Throat, ed. 3, London, Cassell & Company, Ltd., 1926, p. 6.

2. Lack, L., cited by Thomson, St. Clair: Diseases of the Nose and Throat, ed. 3, London, Cassell & Company, Ltd., 1926, p. 7.

3. Proetz, A. W.: Some Intimate Studies of Nasal Function: Their Bearing on Diagnosis and Treatment, Ann. Otol., Rhin. & Laryng. **41**:125 (March) 1932.

These experiments proved that an inspiratory stream of air can be deflected and broken by the shape and position of the nasal aperture and also by any mechanical obstacle or alteration in the lateral and middle walls of the nose.

The various structures which, if abnormal, can deflect these inspiratory air currents may be grouped under the general term "nares." Specifically they are the columella, the anterior portion of the nasal septum, the alar cartilage with lateral and medial crura, the upper lateral cartilages and the small constrictor-dilator muscles of the nose.

The following groups of cases will show that each of these structures plays an important role in the deflection of inspired air and the subsequent changes in nasal physiology.

I. ELONGATED, BEAKED NOSE

A beaked nose shows a marked forward prolongation of the anterior septum and downward projection of the angle of the alar cartilages (fig. 1, *1* to *4*).

The patients present alterations in the physiologic condition of the nose. Their usual complaints are nasal stuffiness, frequent "colds," occasional headaches and dryness of the nose. This pathologic syndrome is due to the mechanical alteration of the air currents caused by the distorted shape of the tip of the nose. The downward projection of the "beaked" tip disrupts the normal circuitous route of the inspiratory current of air, producing eddies and whorls in the nasal passages. Heretofore, the usual intranasal treatments and surgical procedures have afforded little relief to the sufferers.

A simple test to ascertain whether these patients will obtain relief is to stand on the right side of the patient and raise the tip of the nose with the middle finger of the right hand. If the patient experiences easier breathing, one can safely say that the operation will give exactly the same amount of improvement. If there is no improvement in nasal breathing when the tip of the nose is raised, operation is not indicated.

The corrective operation for this condition is simple. It is important to ascertain how high the tip of the nose must be raised in order to obtain the desired result. This knowledge is had by raising the tip of the patient's nose until he attains the maximum relief. The height to which the tip must be raised indicates how much cartilage must be removed.

An intercartilaginous incision is made between the upper lateral and the alar cartilage, intranasally, on one side. A small pair of scissors is then inserted into the opening, and the skin is separated from its adjacent perichrondrium and periosteum up to the glabella. The same procedure is followed on the opposite side. A long straight Joseph knife is then inserted through one incision and passed through the

second incision on the other side, making a common communication, which is carried forward to the columella, the upper border of the septum thus being separated from the skin. Then the separation is carried downward in the fibrous communication, the columella being separated from the anterior border of the nasal septum down to the

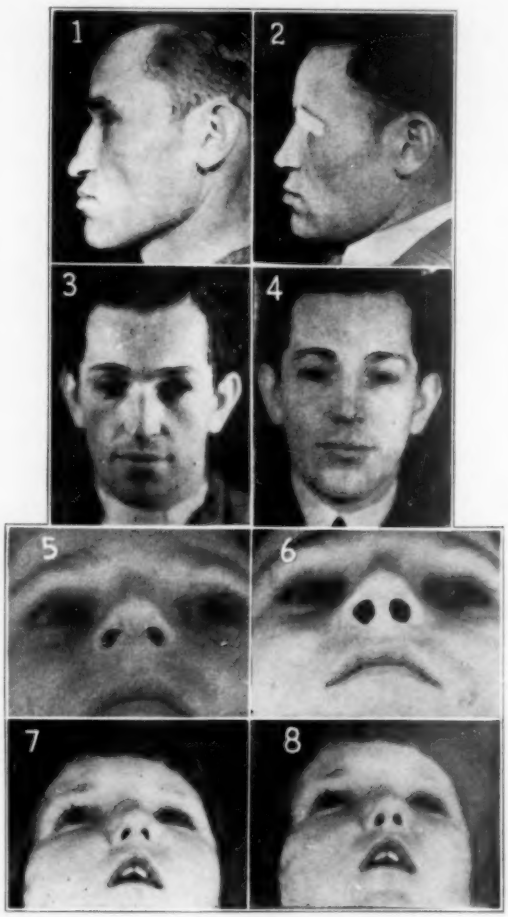

Fig. 1.—Beaked nose, with correction (*1* to *4*). Wide separation of the medial crura of the alar cartilages, with correction (*5* and *6*). Wide columella due only to redundant tissue, with correction (*7* and *8*).

philtrum. A triangle with its base upward and its apex caudalward is removed from the septum with its perichondrium and mucous membrane. The columella and the septum are then sutured together with two orthopedic stitches.

II. HYPERTROPHIED COLUMELLA

Any structural change in the anatomic arrangement of the columella can deflect the inspiratory air current and cause a disturbance in nasal functions. Many interesting abnormalities in this classification may be responsible for such a change.

A. *Wide Separation of the Medial Crura of the Alar Cartilages.*—Normally the medial crus of the alar cartilage comes in close contact with its fellow on the other side, forming a narrow, straight supporting columella. When the medial crura are widely separated, with the interposition of adipose and fibrous tissues (fig. 1, *5* and *6*), the columella becomes broad and short and acts as a buffer to the inspiratory stream of air.

The corrective measure which I have used successfully is as follows: A straight longitudinal incision is made through the center of the columella. The edges are widely separated with fine nasal hooks, an exposure of the anatomic defect being permitted. All the interposing tissue between the medial crura is dissected out thoroughly. The medial crura, now free, are brought together with two heavy black silk mattress sutures. One suture is placed high and the other low on the columella. The longitudinal cutaneous incision on the columella is brought together with fine interrupted dermal sutures. The heavy black silk mattress sutures are allowed to remain for seven days.

B. *Wide Columella Due Only to Redundant Tissue.*—This abnormality (fig. 1, *7* and *8*) is not uncommon in children. The columella is filled with fatty and connective tissue which causes its lower part to flare out, the inlet being thereby narrowed. In such cases, the inspiratory current of air is deflected, nasal function being disturbed. The children present adenoid facies. It will be worth observing these cases in later years, after proper correction of the columella, to see if there are any changes in the facial expressions and characteristics. The oldest operation in this category was done only a little over a year ago, and it is too early to make any definite statement. The patients that have been operated on showed marked improvement in nasal hygiene, mental alertness and general physical condition.

The corrective operation for this condition follows: A longitudinal incision is made, separating the columella from the anterior border of the septum. Both lips of the columella are retracted outward with fine nasal hooks. All redundant tissue in the columella is resected. Histologic examination will show adipose and fibrous tissues, with some muscle. The septal mucosa is gently sutured to the epidermal layer of the columella on both sides. Two or three sutures may be required to bring these structures into alinement. Two heavy black mattress sutures are used to bring together the lips of the columella. This mattress suture

is placed only through one side of the columella to the other. When these sutures are brought together, the columella becomes rather narrow, and the nasal aperture rounds out to its normal shape.

C. *Redundant Tissue of Entire Nares.*—This condition is usually due to cutaneous disorders of the face, especially acne (fig. 2, *1* and

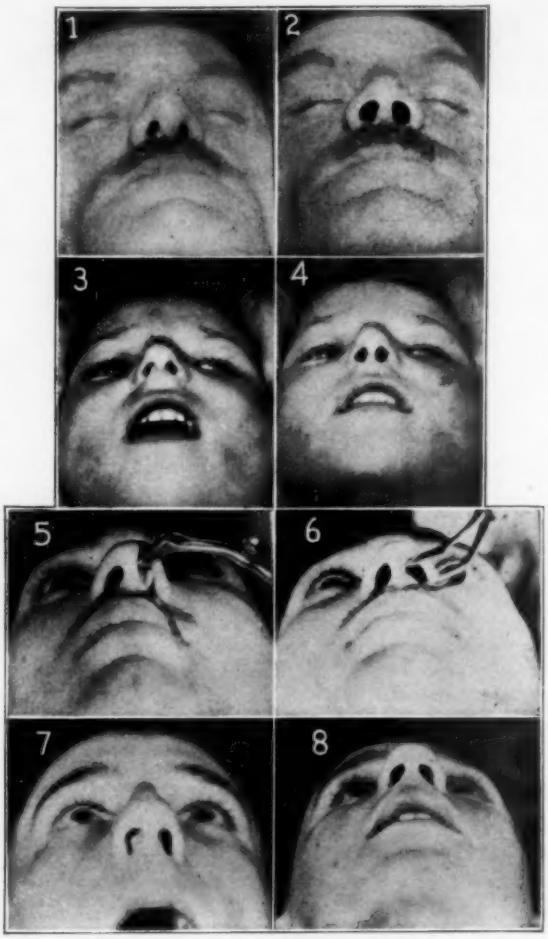

Fig. 2.—Redundant tissue of entire nares, with correction (*1* and *2*). Anterior septal dislocation with hypertrophy of the columella, with correction (*3* and *4*). Dislocation of a fractured septum to the left with concavity of the septum on the right, with correction (*5* and *6*). Dislocation of anterior part of septum, opposite side not requiring adjustment, with correction (*7* and *8*).

2). The redundancy may be so marked that the nasal aperture may become completely occluded.

The corrective surgical procedure in such cases is the operation on the columella just described plus a parallel incision in each prominent

rim of the nostril. Care must be taken that when the redundant tissue from the side of the nose is being dissected out the small nasal muscles and the lateral crus of the alar cartilage are not disturbed. The importance of these small muscles will be discussed later. Dermal sutures are used to close the wound after enough tissue has been removed.

D. *Anterior Septal Dislocation with Hypertrophy of the Columella.*—Patients with this type of abnormality usually present an anterior dislocation of the septum due to fracture. The dislocated segment of the septum is pushed forward into the columella, which is broadened while the nasal entrance is narrowed (fig. 2, *3* and *4*). The right side of the columella in fig. 2, *3*, shows the dislocated segment of the septum, which causes the columella to flare out. The hypertrophy on the left side is due to redundant tissue in the columella.

A longitudinal incision separates the anterior septum from the columella. The lips of the columella are retracted outward with fine nasal hooks. The redundant tissue on the left side is dissected out. The dislocated segment of the septum is dissected out in toto from the columella on the right side. If the structural support of the nose is not disturbed, the dissected segment is not reinserted, and the incisions are closed in the usual manner.

If too much anterior septum has been removed, then the segment is reinserted between the lips of the columella. The manner in which this graft is held together will be described further on.

III. DISLOCATION OF ANTERIOR NASAL SEPTUM DUE TO FRACTURE

Dislocation of the anterior part of the nasal septum due to fracture is seen frequently. The disturbance in the nasal function created by the deformity is well known to the rhinologist. Many classic operations have been described as a remedy. Figure 2, *5*, shows marked dislocation of a fractured septum toward the left, with concavity of the septum on the right side. A longitudinal incision is made, separating the columella from the anterior part of the septum. The septal mucosa and perichondrium are separated from the anterior part of the quadrilateral cartilage on both sides, to a point behind the dislocation. This entire portion is dissected out. As much as possible of the projecting portion of the dislocated part is shaved off with a sharp scalpel. If the cartilage is still bent, the tension can be released with vertical and horizontal incisions through it. It is then reinserted between the open lips of the columella. The septal mucosa is sutured with dermal sutures to the epidermal layers of the columella. It is necessary to suture the graft in place to prevent sloughing or buckling caused by its falling either forward or backward.

Two heavy black silk mattress sutures are placed through the septal portion of the inserted cartilage, carried through to the opposite side

and then through the columella on the same level and united. These supporting sutures remain from seven to ten days, when the "graft" will become well embedded in a mesh of fibrous tissue. The purpose of this procedure is to straighten out the weak concave wall on the opposite side of the dislocation and to give support to the nose. Figure 2, *6*, shows the end result. Figure 2, *7*, shows a marked dislocation of the anterior part of the septum, but the opposite side does not require any adjustment. The classic procedure of removing the dislocated segment in toto without interfering with the cartilage and its coverings on the opposite side is well known and requires only a comment. It is best to remove a small portion of the mucous membrane when bringing together the two open edges of the original incision. If this is not done, the excess areas of mucous membrane will buckle on each other and act as a buffer to the inspiratory stream of air.

IV. HYPERTROPHIC UPPER LATERAL CARTILAGES

Hypertrophy of the upper lateral cartilages is not uncommon. It may be congenital or acquired through trauma. Its position is usually downward and mediad. This prevents the inspiratory air from taking its normal circuitous route and gives rise to nasal symptoms.

If the lower lateral cartilage is atrophied, the downward pressure of the hypertrophic upper lateral cartilage usually causes an inward pinch on the lateral part of the lower third of the nose. In such cases, the surgical procedure is to free the lateral extremity of the upper lateral cartilage from its fibrous connection to the apertura piriformis, leaving its medial attachment to the upper border of the septum nasi intact. The freed end is then brought downward, filling in the weak area of the nose between the skin and the nasal mucosa, held in place with sutures endonasally, or through the nose, and sutured on the outside, with an interposed layer of cotton or gauze.

This technic accomplishes two things: First, it strengthens the weak lateral wall of the nose, and, second, it eliminates the endonasal obstruction and permits the inspiratory current of air to assume its normal route.

In cases in which the lower lateral cartilage is not atrophied, a submucous resection of the hypertrophied tissue is sufficient to allow the inspiratory current of air to have ample space.

V. MUSCULAR IMBALANCE OR MUSCULAR ATROPHY OF THE NARES

The muscles of the nares work antagonistically. There are two sets of nasal muscular fibers, the constrictors and the dilators. These muscles are considered rudimentary. However, I do not subscribe to that theory. These muscles have a definite function, and when their

contractile powers are lost there is a marked alteration in nasal function. In a forthcoming paper, the origin, insertion and definite function of each muscle of the nares will be described, as well as the atrophy of the muscles of collapsed nares.

Such cases are observed frequently. On inspiration there is marked collapse of the entire lateral wall of the nose, in some instances giving a slitlike appearance to the nostrils. The majority of patients, however, do not have so marked a collapse. Because of the weak and atrophied nasal muscles, the area corresponding to the upper lateral cartilage is constricted on inspiration, although the rim of the naris flares out. There is a sense of obstruction, and the patient inspires more forcibly in an attempt to bring more air into the nares. This effort causes marked constriction of the weakened area, which at times impinges on the nasal septum, producing more obstruction to the inspiratory current of air.

Fig. 3, *1* to *4*, illustrates the cases of complete muscular imbalance. On inspiration there is a collapse of the entire nose, only a small distorted stream of inspiratory air being permitted to enter. The alteration in nasal function in such conditions is marked.

Figure 3, *1* and *2*, presents a patient with an unusual symptom, anosmia. She perceived only the strongest odors. Directly after operation she was able to detect the odor of a rose. There is a physiologic explanation for such a change. For one to become conscious of odors there must be some stimulation of the protoplasmic filaments in the olfactory vesicle of van der Stricht. The olfactory hairs in this region act as receptors and respond to the stimulation carried to them by the inspiratory current of air. The reason that many patients with this condition have anosmia with nasal stuffiness and frontal headaches is that the inspiratory current of air becomes wholly or in part deflected from its normal path.

Figure 3, *5* and *7*, shows patients in whom there is not a complete collapse of the nares on inspiration but a constriction of the upper lateral portion (corresponding to the upper lateral cartilage), caused by imbalance of muscular contraction or atrophy of the upper lateral cartilages. This constriction in many cases is so marked as to cause impingement on the septum, but the lower part of the nostril (inlet) remains open. Photographically, it is not possible to show the constriction of the upper lateral part of the nares toward the septum on inspiration. Figure 3, *6* and *8*, shows the correction of this condition, with the subsequent increase in the length and diameter of the nares.

In this group, a simple test will indicate whether surgical intervention will be beneficial. A small thin wooden applicator is inserted in each nostril. The nares are pushed outward, upward and laterally as far as

possible. If the patient experiences a marked increase in the volume of air on inspiration, one can safely predict that the operation will afford the same amount of relief.

The surgical correction of this condition is simply to transpose the nares more laterally in a fixed position, so that they will not collapse on

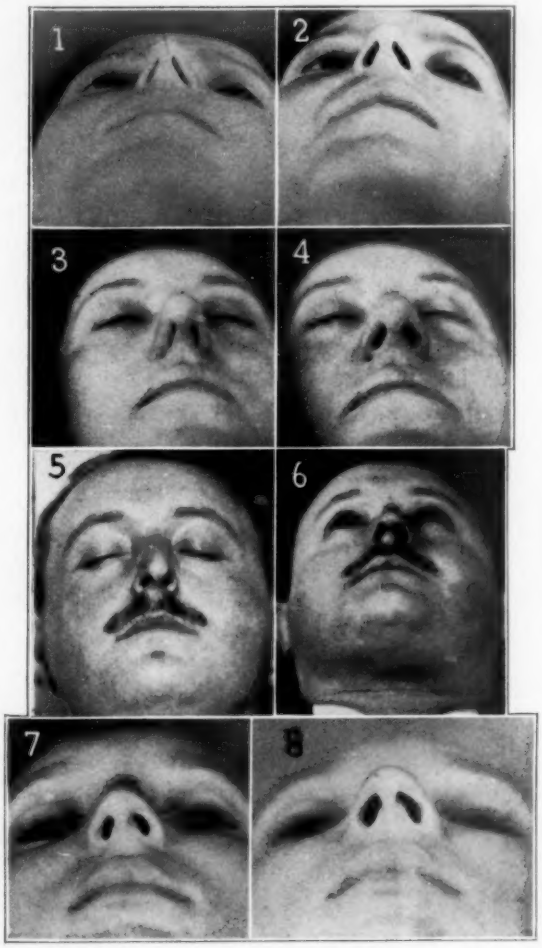

Fig. 3.—Complete muscular imbalance, with correction (*1* to *4*). Constriction of the upper lateral portion of the nares, with correction (*5* to *8*).

inspiration. This operation has been termed "lateral transfixation of the nares" (fig. 4, *1* to *12*).

It is performed with the patient under local anesthesia. Figure 4, *1*, shows a semilunar incision in the nasolabial fold. Figure 4, *2*, shows an Allis clamp grasping the upper edge of the cutaneous incision, with a

scalpel dissecting the deeper layers. Figure 4, *3*, shows all the layers, viz., cutaneous, subcutaneous and muscular layers, totally resected. The thin layer exposed and left intact is the mucous membrane of the nose. Figure 4, *4*, shows a long, straight needle with 00 forty day chromic

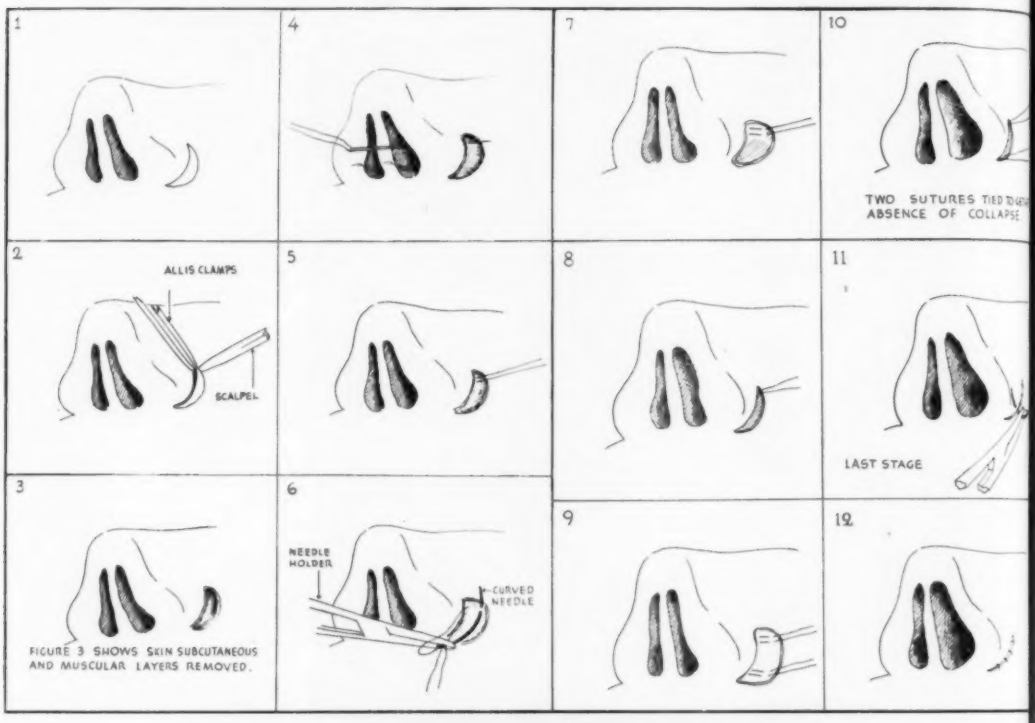

Fig. 4.—Lateral transfixation of the nares (*1* to *12*). Anterior atresia of the nares, with correction (*13* and *14*). Acquired collapse of the tip of the nose, shortening and narrowing the nostrils, with correction (*15* and *16*).

catgut being passed through the nasal mucosa at a level corresponding to the highest point of the incision and carried outward. Figure 4, *5*, is the same procedure with the same suture directly below it. Figure 4, *6*, shows the upper suture on a curved needle being fixed laterally on

the same plane. Figure 4, *7,* shows these two sutures completed but untied, with the entire nares collapsed. Figure 4, *8,* shows these two sutures tied and fixed laterally, with the upper part of the nares wide open and enlarged. Figure 4, *9* and *10,* shows the same procedures but at the lower end of the incision and also the relation of the nares to the sutures when tied and untied. Figure 4, *10,* shows that when the sutures are tied the collapsed nares become enlarged in height and width and become permanently transfixed laterally. Figure 4, *11,* shows the dermal sutures closing the cutaneous wound, and *12,* the final result.

No packing is inserted in the nose. No external dressing whatever is employed. The nose is kept clean with sterile glycerin. There is absolutely no change in the external contour of the nose, and the scar is not visible, as it is well covered in the new nasolabial fold. The dermal sutures may be removed on the third day.

VI. SMALL NOSTRILS

Small nostrils present a distinct obstruction to the inspiratory current of air and are easily diagnosed. The advent of rhinoplastic surgery has produced more anterior nasal atresia than any other etiologic factor. This atresia is formed when an inexperienced operator attempts to narrow the tip of the nose and unwittingly removes too much nasal mucosa. Naturally there are many other etiologic factors, Figure 4, *13* and *14,* shows anterior atresia of the nares, with correction. The weblike atresia was removed in toto, and a Thiersch graft was inserted.

There is another group of cases which interests the rhinologist. The tip of the nose, as it collapses, shortens and narrows the nostrils. This condition may be congenital or acquired. When acquired (fig. 4, *15*) it is sometimes due to a high and anterior submucous resection.

The method of correction varies with the individual case. If the columella is weak, an ivory or cartilaginous support placed within its lips from the tip of the nose to the philtrum may be sufficient. If the nares are weak or collapsed, this support is combined with lateral transfixation of the nares, as illustrated in figure 4, *16.*

SUMMARY

Physiologists in the past have shown that the inspiratory and expiratory streams of air take a definite circuitous path in the nasal chamber.

These observations were further established by the works of Proetz, using litmus paper and smoke trails.

Proetz showed in his experiments that the direction of the air currents is determined by the shape and position of the inlet (nostril). He further showed that when obstacles of any description are introduced either on the septal or the lateral wall, they produce the most pronounced changes, and the current is broken into eddies and whorls.

Observations in a variety of cases have been described which corroborate these experiments in every detail.

Although not enough time has elapsed to warrant dogmatic statements, the following observations have been made: After surgical correction the nasal breathing improved from 50 to 100 per cent. The number and intensity of colds will be reduced. In selected cases, frontal headaches were relieved and anosmia cured. Postnasal drip diminshed 30 per cent in some cases. In the majority of the patients mental alertness and physical condition were markedly improved.

I intend to show in a subsequent paper that the so-called rudimentary muscles of the nose have a definite physiologic role and that failure of these muscles to function produces a marked disturbance in nasal hygiene.

A new operation, "lateral transfixation of nares," is presented.

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A METHOD FOR RECONSTRUCTION OF POSTAURICULAR DEFECTS

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BROOKLYN
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OMAHA

There falls into the hands of every reconstructive surgeon a certain group of cases in which postauricular defects have been operated on repeatedly with little or no success. These defects may be the result of uncontrolled infection, of incomplete operation on the mastoid, such as simple antrotomy and poorly executed radical mastoidectomy, or, as in one case which we observed, of the spontaneous perforation of an untreated diseased mastoid.

The resulting unsightly defect is manifested as a depression in the region of the former mastoid prominence, covered wholly or in part by a thin, reddened and contracted scar, which is usually painful in varying degrees and is further made an annoyance by a discharge.

With the demands on the modern plastic surgeon becoming more and more exacting, the need was felt for a review of this problem. The anatomic and surgical questions presented were reconsidered in order to establish some operative principles to insure, as far as possible, satisfactory functional and cosmetic results.

REVIEW OF THE LITERATURE

Zoufal [1] in his description of the first radical mastoidectomies considered a permanent postauricular opening essential. This was disfiguring. Epithelization was allowed to take place from behind the ear rather than encouraged by splitting the membranous external auditory meatus after immediate closure of the wound, as is now commonly done. The endaural approach, revived by Lempert,[2] gives excellent results from the cosmetic point of view.

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1. Cited by Gerber, P. H.: Handatlas der Operationen am Schläfenbein, Wiesbaden, J. F. Bergmann, 1904.

2. Lempert, J.: Endaural, Antauricular Surgical Approach to Temporal Bone: Principles Involved in This New Approach; Summary Report of One Thousand, Seven Hundred and Eighty Cases, Arch. Otolaryng. **27**:555 (May) 1938.

The successful correction of postauricular defects depends on the the use of various types of tissue grafts and flaps. Simple undermining of the skin adjacent to the defect to obtain relaxation, with accurate opposition of its edges, is the most common method of secondary closure. The skin is frequently under tension after this type of operation, but the defect is usually small, and successful closure is frequently obtained.

The simple epidermal flap described by Mostig-Moorhof [1] is cut from below the defect, retaining an attachment at its inferior periphery. The flap is liberated and rotated into the cavity with the epithelium toward it. Slight modifications of this technic, with the flaps placed posterior or superior to the defect, with or without relaxing incisions, were described by Trautmann,[3] Beck,[4] Goldstein,[4] Kerrison,[4] Watson,[5] Lautenschlager [4] and Heine.[6]

The periosteal and subcutaneous flap was advised by Popper,[7] and in describing his technic he suggested the use of a large horseshoe-shaped periosteal flap cut from behind the mastoid and attached to the postauricular fold, which is reflected into the defect. McNichols [8] and Ashley [9] also suggested periosteal and subcutaneous flaps, variously modified, for the closure of such defects.

Copps and McCormick [10] use subcutaneous tissue posterior to the defect for filling the concavity of the mastoid, but they do not incise further than the initial incision for freeing the skin. Instead, they leave an area over which the skin is placed under tension, without subcutaneous tissue in its posterior portion but with skin and the transplanted subcutaneous tissue over the defect.

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3. Trautmann, cited by Phillips, W. C.: Diseases of the Ear, Nose and Throat, Medical and Surgical, ed. 4, Philadelphia, F. A. Davis Company, 1917, p. 303.

4. Cited by Kirschner, M.: Operative Surgery, Philadelphia, J. B. Lippincott Company, 1937, p. 39.

5. Watson, D.: Correction of Mastoid Defects, Proc. Roy. Soc. Med. **23**: 397, 1930.

6. Heine, B., cited by Kerrison, P. D.: Diseases of the Ear, ed. 2, Philadelphia, J. B. Lippincott Company, 1921, p. 459.

7. Popper, O.: The Use of Periosteal Flap Grafts in Mastoid Operations, J. Laryng. & Otol. **47**:126, 1932.

8. McNichols, W. A.: Prevention of Postauricular Fistula by Periosteal and Subcutaneous Tissue Flaps, Ann. Otol., Rhin. & Laryng. **45**:475, 1936.

9. Ashley, R. E.: A New Method for Closing a Postauricular Fistula, Ann. Otol., Rhin. & Laryng. **46**:477, 1937.

10. Copps, L. A., and McCormick, G. L.: The Closure of Persistent Postoperative Mastoid Fistula with a Subcutaneous Pedunculated Flap, Arch. Otolaryng. **27**:472 (April) 1938.

A pedicle scalp flap is employed by Straatsma [11] for filling postauricular defects. On the raw surface of the flap is placed a Thiersch graft. After the flap with its accompanying graft has taken in its new bed, its unusual portion is returned to its original site, and a split skin graft from the thigh or abdomen is used as necessary for covering the remaining defect.

Ersner and Myers [12] utilize a "racket-shaped" pedicle flap from the neck, which is transplanted into the defect, with its pedicle severed after a take is assured.

Straatsma and Peer [13] reported the successful use of free grafts of fat from the thigh and abdomen for the correction of postauricular defects. Eagleton [14] employed bone and bone chips for the same purpose.

A purse string suture is used by Passow [4] for closing the deep layer of a double layer flap made over the same type of irregularity.

Kisch [15] described a flap from the temporal muscle, in which a "tongue" of muscle is transferred directly into the defect, retaining an attachment in its anterior portion. Almour [16] described a similar operation, in which the muscle is freed and transposed directly into the defect, retaining an attachment "by a narrow pedicle located in the upper anterior quadrant of the mastoid cavity." The flap is rotated on its pedicle to have the periosteum next to the bone, while the operation of Kisch does not call for such a procedure.

FACTORS IN THE CHOICE OF OPERATION

The incidence of large postauricular deformities following operation on the mastoid is increased by such concomitant systemic conditions as tuberculosis, syphilis, diabetes mellitus, scarlet fever and hematic dyscrasia. Extensive pneumatization of the mastoids, with large cavities resulting from the removal of the diseased bone, usually causes a gross deformity. Poor surgical technic at the time of operation, with failure to appose the edges of the skin properly, or undue tension of the parts tends to minimize the possibilities of a satisfactory result.

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11. Straatsma, C. R.: Repair of Postauricular Fistula Following Radical Mastoidectomy, Arch. Otolaryng. **19**:616 (May) 1934.

12. Ersner, M. S., and Myers, D.: Variation of Pedicle Flap for Epitheliation of Radical Mastoidectomy Cavity, Arch. Otolaryng. **23**:469 (April) 1936.

13. Straatsma, C. R., and Peer, L. A.: Repair of Postauricular Fistula by Means of Free Fat Graft, Arch. Otolaryng. **15**:620 (April) 1932.

14. Eagleton, W. P.: Reconstruction of Mastoid Wound Cavity by Use of Bone Grafts and Chips, Laryngoscope **29**:272, 1919.

15. Kisch, H.: Temporal Muscle Flap, J. Laryng. & Otol. **43**:856, 1928.

16. Almour, R.: Method for Repair of Persistent Postauricular Openings, Laryngoscope **40**:799, 1930.

Secondary infections resulting in erysipelas or an erysipeloid reaction, with which closure is delayed or is not attempted, terminate in the defect being filled with granulations and epithelization taking place in an unsatisfactory manner. It has been found, in reviewing the cases on which this paper is based, that a large portion of extensive postauricular defects follow such complications.

As in all operations involving the skin, after-care is as important as the operation itself. Poorly placed drains and packing, with resulting undue pressure on the epidermis is followed by ischemia and death of the superficial protective covering. The injudicious use of caustics also may contribute to poor recovery.

Allowing the wound created to close superficially before the underlying tissues have filled in the defect is usually followed by unsatisfactory results. As elsewhere in the body, due respect for the skin is demanded if a favorable end result is to be obtained.

With a postauricular deformity resulting from one or more of these factors, it is evident that the first step toward recovery is to eliminate all possible infection. This may entail a revision of the operation on the mastoid.

With a deformity of such a size that clinical judgment dictates the use of some form of plastic procedure, the type of operation must be weighed in the light of its feasibility in the case at hand.

Small defects can frequently be closed by simply undermining the edges of the skin for relaxation, freshening and approximating them.

A simple skin graft or epidermal flap might be indicated if the cavity presents the possibility of an adequate blood supply on a healthy granulating base. This type of repair merely covers the denuded bone and does not restore contour.

A free graft of fat or muscle may be considered. As a prerequisite to this operation a good blood supply plus adequate skin to cover the restored contour without undue tension is mandatory. Grafts of this type have the unfortunate tendency to diminish in size all too frequently, and what appears a good restoration of contour after the operation may literally melt away entirely in a short time.

Bone grafts and bone chips need not be considered in this discussion, as they are doomed to be lost unless they have the most favorable of fields.

A pedicle flap of skin or scalp may be considered. Such flaps carry their own blood supply, but the scar created by the operation is often as unsightly as the original deformity.

The periosteal and subcutaneous flap has definite indications. Nutrition is supplied from the donor site and the scarring is minimal. However, this type of operation is limited to the smaller deformities because

of the nature of the flap itself. The structure of the region from which it is taken precludes the use of too much tissue, because necrosis and ulceration intervene if the blood supply is inadequate.

For large defects, contour must be restored by an operation that itself does not have too great a defect, and the operation chosen must offer a reasonable expectation of success. To do this the following requirements must be met. There must be enough skin to cover the defect completely. The area in some cases is several square centimeters. The base on which the skin is to rest must carry its own blood supply and be such that postoperative absorption does not destroy the contour achieved. If the contour is restored with a tissue having an adequate blood supply and enough available skin to make an apposition of the various layers of tissue, then a favorable termination may be contemplated. To fulfil these conditions a muscle flap is the only material available.

ANATOMY

With these requirements, the anatomy of the temporal muscle was reconsidered when it was found that the middle temporal artery has subdivisions that might be utilized in the construction of a muscle flap.

The middle temporal artery is a branch of the superficial temporal artery arising just above the zygoma, and after perforating the temporal aponeurosis and muscle it ascends through the substance of the temporal muscle itself.[17] In anatomic literature the information as to the exact blood supply of the temporal muscle is meager, so that a study of the problem was found necessary. Human material consisting of both injected and uninjected specimens was examined, and it was found that the middle temporal artery usually branches into two divisions, anterior and posterior. The posterior division supplies the most posterior and inferior portion of the temporal muscle as its fibers pass directly posteriorly and horizontally.

In the series of specimens which we examined, the posterior division of the middle temporal artery was found to be fairly large, lying somewhere within 1.5 cm. of the inferior-posterior portion of the muscle. In 2 cases the artery was found to subdivide still further just as it was given off the middle temporal artery, with the result that two small arteries were present in the area rather than one relatively large branch. The posterior branch of the middle temporal artery has its origin anterior to the superficial temporal artery and then turns medially and posteriorly, to supply the muscle to be utilized in the construction of the flap. The muscle may be used as far anterior as the region where the temporal

17. Piersol, G. A.: Human Anatomy, ed. 9, Philadelphia, J. B. Lippincott Company, 1930.

artery crosses its fibers, in the knowledge that it still has a good blood supply up to this point.

To satisfy the requirements further, a flap has to be constructed in such a manner that the lumen of the posterior branch of the middle temporal artery will not be occluded by torsion and at the same time have a broad base to insure the presence of this artery in it.

DESCRIPTION OF THE OPERATION

The following operation was devised in an attempt to fulfil the requirements laid down.

The incision starts at a point between the superior temporal line and the sagittal suture, about 2 cm. anterior to the biauricular line (fig., *A*) and is carried down to the temporal fascia but does not involve it. It is straight, crossing the biauricular line several centimeters above the ear, skirting the postauricular defect anteriorly and posteriorly and extending over the region of the mastoid.

The epithelial remnants within the anterior and posterior incisions over the defect are completely removed. An attempt is not made to remove granulation tissue. The defect is then packed temporarily, as it is ready to receive the muscle flap.

The skin is undermined anteriorly and posteriorly from the edges of the incision, with the line of cleavage on the temporal fascia. The undermining is carried cephalad beyond the superior temporal line, posteriorly to the occipital region, so that the most posterior portion of the temporal muscle is well exposed. Inferiorly and anteriorly the skin is freed as far as the zygomatic arch. It is liberated 3 to 5 cm. below the tip of the mastoid. This procedure may seem a bit radical, but it is essential if there is to be enough skin to allow proper closure of the large defects for which the operation is intended. Considerable bleeding will be encountered under the flap raised in this manner, but this should cause no concern, as it can be adequately controlled by placing warm moist packs well underneath the flaps and applying manual pressure over them.

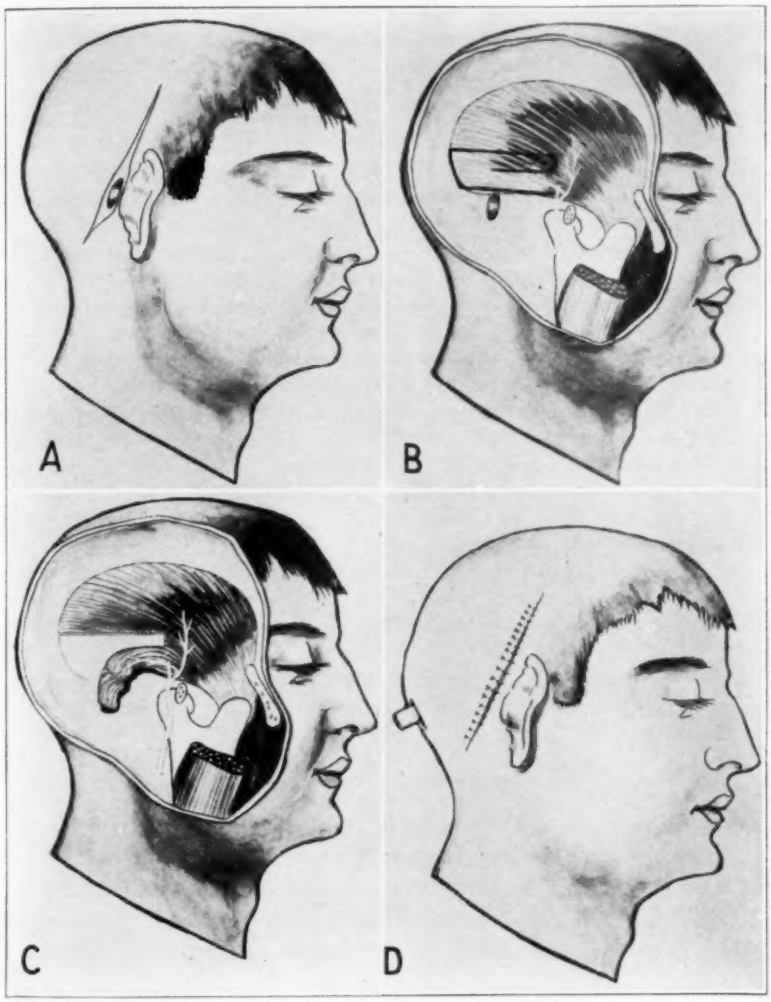
After this is done the actual construction of the muscle flap is started. The temporal muscle is clearly exposed in its posterior inferior origin. The fibers should be clearly visible as far anteriorly as a point directly superior to the tragus. An incision is next made through the fascia and the muscle down to the bone, extending from the most posterior-inferior portion of the muscle horizontally forward to a point directly above the anterior-superior attachment of the ear. This will be the inferior portion of the muscle flap.

Another incision is now made from 2 to 5 cm. above and parallel to the original incision. This too is carried directly down to the bone and reaches anteriorly to a point directly above the forward tip of the first incision.

Next the muscle between the two incisions is freed from the underlying bone as far anteriorly as the incisions extend and posteriorly well toward the superior temporal line, care being taken not to free the flap. At a point midway between the two incisions, in their anterior aspect, still another incision is made parallel to the original one. This is carried posteriorly to a point at least midway in the flap, the actual extent depending largely on the size of the defect to be filled.

At this stage the posterior division of the middle temporal artery should be contained within the lower division of the muscle flap (fig., *B*).

The muscle flap is cut completely free from its posterior attachment, and the amount of lengthening needed is estimated. The extent of splitting of the flap is determined by the actual amount of tissue necessary to restore contour. The superior-anterior portion of the flap is next cut free, and it will be found that by



A, direction and extent of the incision. The epithelial remnants are completely removed in the region of the defect. *B,* outline of the muscle flap, showing the contained posterior division of the middle temporal artery. The superficial temporal artery is shown only at its margin. *C,* flap liberated and rotated into the region of the defect, carrying with it the artery. *D,* complete closure of the initial incision, with a drain in the dependent portion of the scalp flap.

rotating this portion of the muscle it can be made to reach the defect even though there is some distance between the attachment of the flap and the defect (fig., *C*). It is so rotated as not to impair its blood supply.

The periosteal portion of the flap is allowed to remain on the surface instead of an attempt being made to twist it so that it fits next to the bone. The work of Levander [18] is cited to substantiate our impression that the periosteum per se is not important in the procedure. The flap is not further incised nor mutilated in any manner that may cause excessive scarring, which might increase the possibilities of absorption.

The flap is fitted into its new position with the contour slightly overcorrected. If the correction is for a defect following radical mastoidectomy the muscle is made to fill the defect well into the middle ear, but the lumen of the canal is maintained by means of a gauze pack inserted through the external auditory meatus. The muscle flap is firmly packed against this. The muscle itself is anchored so that there are no "dead spaces," the fascia over the muscle being utilized and sutures being passed through it into the fascia and periosteum in the surrounding area.

The primary incision is closed tightly with no drains, and every attempt is made to get satisfactory apposition of the skin in the approved manner. A stab incision is made well posteriorly and in the most dependent portion of the liberated flaps (fig., *D*). This is well within the hair line, and in this site a plain rubber drain is inserted to allow the escape of blood from any hemorrhage under the flaps. This leaves only a small scar that will eventually be covered with hair.

A model of the ear, in two parts, is made of dental compound. The anatomic position of the ear must be preserved and the postauricular sulcus maintained. The model is also fitted into the external auditory canal and made to conform to the uneven contour of the ear. This is in turn smoothed over on the surface, so that a large dressing may be applied, exerting pressure equally over the ear, the postauricular defect and the flaps in their entire extent.

The presence of a small pack to maintain the lumen of the external canal in cases of repair following radical mastoidectomy is most important. A wick saturated with a 70 per cent solution of alcohol has been employed with success for this purpose.

The rather complicated dressing is justified because the pressure, which is of paramount importance, prevents the cartilages of the ear from being forced into the region of the graft.

EXPERIENCE WITH THE OPERATION

In three years this operation has been successfully employed in 15 cases. All the patients had been previously operated on one or more times, and 1 had had eleven unsuccessful attempts at closure.

In this series of cases no infection has been encountered to cause a breaking down of the wound, and, as before stated, this is attributed largely to the freedom of the skin flaps and to the proper preparation of the muscle flap with its contained artery. In no case was it found necessary to make a relaxing incision for proper closure.

SUMMARY

1. A brief review of the literature pertaining to the procedures for the correction of postauricular defects is given.

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18. Levander, G.: A Study in Bone Regeneration, Surg., Gynec. & Obst. **67**: 705, 1938.

2. A more detailed study of the blood supply of the temporal muscle, as found in injected and in uninjected human specimens, is described. The posterior division of the middle temporal branch of the superficial temporal artery is found to be in a position to allow the construction of a flap from the temporal muscle carrying a dependable blood supply for the correction of postauricular defects.

3. A "radical" operation for the closure of large postauricular defects is described in detail. This operation is based on a series of 15 cases, in all of which repair was successfully performed by this method after one or more previous unsatisfactory attempts at closure by one or several of the conservative methods described.

NATURE OF VITAMIN B AND ITS COMPONENTS

WITH SPECIAL REFERENCE TO NERVE DEAFNESS

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The accumulated total of human knowledge is incomprehensible. A few hundred years ago an aspiring scientist took all knowledge as his province, but the ever mounting mass of information necessarily led to specialization. The inevitable penalty of working in a restricted field is ignorance of progress outside one's special domain until some significant overlapping necessitates inquisitive excursions abroad. The possible relation between deficiency of vitamin B and at least some forms of nerve deafness, pointed out by Selfridge,[1] and an attempt to alleviate a condition heretofore largely intractable entail certain questions which must be answered if therapeusis is to have some semblance of rationality. What is the chemical and biologic nature of this complex catalyst? Can a symptomatic picture be formulated so that the need for it can be diagnosed clinically? Are there any laboratory procedures to aid investigators? What are the meanings of the new names and standards of measurement occurring in published reports? To answer these and other questions extensive search of the literature through the 1937 volume of the *Quarterly Cumulative Index Medicus* was undertaken. The information on vitamins, particularly vitamin B, was found to be in a chaotic state of flux and so voluminous that a really complete survey would be impossible. The following paper is written in the hope that the material here presented will obviate the necessity of much collateral reading on the part of others.

HISTORICAL DATA

Beriberi was known to the Chinese in 2600 B. C.,[2] and, of course, scurvy was the greatest hindrance to maritime exploration in the great era of geographic expansion. It is interesting that the celebrated Cook

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Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, May 1939.

1. Selfridge, G.: Eighth Nerve High Tone Deafness from the Nutritional Standpoint, Ann. Otol., Rhin. & Laryng. **46**:93-118 (March) 1937.

2. Rose, M. S.: The Foundations of Nutrition, New York, The Macmillan Company, 1938.

attributed the latter to dietary faults, and a Japanese naval officer in 1872 reduced the incidence of the former by an improved dietary regimen. In 1881 N. Lunin [3] postulated the vitamin theory on his observation that proteins, fats, carbohydrates and mineral substances were insufficient to support life. Eijkman in 1897 concluded that beriberi is a disease produced by the toxicity of polished rice, but Grijns in 1901 arrived at the conception that a deficiency is at fault. According to Booth and Hansen,[4] "Funk in 1912 proposed the name 'vitamin' for the substance derived from rice polishings which cured beri-beri." Independently McCollum and Davis in this country (in 1914) found a water-soluble substance necessary for growth which proved to be identical with Funks'. This water-soluble substance was termed vitamin B in distinction to the fat-soluble element called vitamin A. Through conflicting biologic experiments it soon became apparent that B is not an entity but a collection of compounds with various chemical and biologic properties.[5]

NATURE OF VITAMIN B

At the present time the following subdivisions of the B group are recognized.[6]

B_1, thiamin [7] (aneurin or torulin), is the antineuritic vitamin. It has been extracted in pure form from natural sources and has been synthesized. It is a complex sulfur, nitrogen and carbon ring compound and forms with hydrochloric acid a white crystalline solid, which is water soluble.[4] It is relatively heat resistant when dry but is rapidly destroyed at 100 C. when moist, especially in neutral or alkaline mediums. In acid mediums it is much less affected by heat.[7a]

Its particular biochemical activity [8] is as an essential catalyst in the combustion of carbohydrate, the oxidation being carried to the lactic acid–pyruvic acid stage and there arrested. Pyruvic acid does not appear

3. Cited by Addinall.[10b]

4. Booth, M., and Hansen, A. E.: Present Day Status of Vitamins: Review, Journal-Lancet **57**:530-544 (Dec.) 1937.

5. Jones, D. B.: A Summary of Our Present Knowledge of Vitamins, United States Department of Agriculture, Bureau of Chemistry and Soils, September 1932. Mendel, L. B., and others: The Vitamins: A Symposium on the Present Status of the Knowledge of Vitamins, Chicago, American Medical Association, 1932.

6. (*a*) Peters, R. A.: Vitamin B Complex, Brit. M. J. **2**:903-905 (Nov. 7) 1936. (*b*) Lepkovsky, S.; Jukes, T. H., and Krause, M. E.: Multiple Nature of Third Factor of Vitamin B Complex, J. Biol. Chem. **115**:557-566 (Sept.) 1936.

7. (*a*) Williams, R. R.: The Chemistry of Thiamin (Vitamin B_1), J. A. M. A. **110**:727-732 (March 5) 1938. (*b*) Riboflavin, the Accepted Name for Vitamin B_2, report of the Council on Pharmacy and Chemistry, ibid. **108**:1340-1341 (April 17) 1937.

8. Peters, R. A.: Biochemical Lesion in Vitamin B_1 Deficiency, Lancet **1**:1161-1165 (May 23) 1936.

in normal brain tissue but is found in such tissues of animals suffering from a deficiency of B_1 and in large quantities also in the blood of patients with beriberi. The addition of B_1 to the brain tissue in vitro causes a continuation of the combustion. This is the only evidence in vitro of the action of vitamins.

Thiamin is stored in the liver, kidneys and brain, but not for long, and is eliminated in the urine and feces.[9] It is not toxic when ingested in large quantities.[7a]

The term B complex refers to all the water-soluble vitamins together and therefore does not designate a chemical entity. B_2 (in England), or G (in the United States), is riboflavin ($C_{17}H_{20}N_4O_6$) (not to be confused with flavones, which are allied to plant pigments or with such substances as acriflavine) and is related to the purines, pyramidines and nucleotides. It is identical with the yellow oxidation enzyme of Warburg. It is destroyed by visible light,[4] especially the blue-violet portion, and by alkalis and is fluorescent in the presence of ultraviolet radiations. It is soluble in water, insoluble in alcohol and heat stable. It rotates the plane of polarized light. It has been obtained pure both from natural sources and by synthesis (Enler, 1934 [10]) and cannot be synthesized in the animal body. It has been demonstrated to unite with a protein to form an enzyme. Its biochemistry has not been elucidated, but it is necessary for life, and its absence is probably not the cause of human pellagra.

The remaining fractions are characterized chiefly by the symptoms produced by their absence from the diets of experimental laboratory animals, little being known of their chemical nature:

B_3, the existence of which is doubtful,[6a] is said to be necessary for growth in pigeons. Its necessity for man has not been established.[11]

B_5 is usually described with B_3, and between them they include the chicken antipellagra factor. B_5 is alkali and heat stable and is necessary for the nutrition of pigeons [12] but is of questionable status.[11]

B_4 also is obscure.[6a] It is perhaps a variation of B_1.[4] Its absence causes paralysis, encephalomalacia and ataxia in animals. It is water soluble, labile and easily destroyed by oxygen.[12] It is not abundant in foods.[11]

B_6 is also known as H, or the Y factor of György.[13] It is usually termed the rat antidermatitis fraction.[6a] Its absence causes a specific

9. Gutman, J.: Modern Views on Vitamins and Treatment of Nutritional Disorders, M. Rec. **146**:129 (Aug. 4); 228 (Sept. 1) 1937.

10. Enler, cited by Rose.[2]

11. Wilder, R. M., and Wilbur, D. L.: Diseases of Metabolism and Nutrition: Review of Certain Recent Contributions, Arch. Int. Med. **59**:512-555 (March) 1937.

12. Elvehjem, C. A.: Present Status of Vitamin B Complex, Am. J. Pub. Health **25**:1334-1339 (Dec.) 1935.

13. Cited by Schultz, H. W., and Mattill, H. A.: Studies on Vitamin B Complex, J. Biol. Chem. **122**:183-198 (Dec.) 1937.

disease in rats characterized by dermatitis of the ears, nose and paws, a bloody mouth and a spectacled loss of hair around the eyes. It is relatively stable to heat and alkali. It is found in liver with vitamin B_1 but occurs in fish and cereals, which are poor in B_1. Deficiency of it may be related to pernicious anemia and agranulocytosis in rats.[1]

The following also are mentioned: the P-P. fraction, the human anti-pellagra fraction, or nicotinic acid;[4] the dog black tongue fraction; the pigeon heart block fraction; a catalyst for growth of molds and micro-organisms; factor 2, an essential for growth, equivalent to the precipitate factor of Elvehjem, Koehn and Olson [13] and probably identical with one of those previously mentioned, and vitamin E, deficiency of which is related to sterility in rats, which may possibly be a component of the B complex.[2]

From a practical standpoint, then, one has to deal with vitamin B_1, or thiamin; G (B_2), or riboflavin; nicotinic acid; vitamin B_6, which has been isolated but not identified, and a group of less well known substances which comprise the remainder of the complex.

SYMPTOMS OF DEFICIENCY OF VITAMIN B

Vitamin B_1.—In laboratory animals (rat and pigeon) absence of B_1 leads to anorexia, loss of weight, polyneuritis, peculiar convulsions and a specific bradycardia in rats,[6a] due to the toxic effect of pyruvic acid on the cardiac muscle.[14] The vitamin is necessary for growth in the young and health in adults. Disturbance of intestinal function, failure of lactation and atrophy of the gonads are reported.[4]

Severe deficiency of B_1 in man causes beriberi,[4] of which the principal symptoms are as follows:

(*a*) Peripheral neuritis, with paralysis of the extremities, muscular atrophy and edema.

(*b*) Vasomotor symptoms, i. e., palpitation of the heart, dyspnea and enlargement of the right side of the heart.

Observed symptoms in experimental B deficiency as reported by Elsom [15] are as follows (symptoms reported by others are interpolated and the reference given):

1. Gastrointestinal symptoms include loss of appetite, the most frequently mentioned symptom;[15] sore tongue; heart burn, and abdominal fulness, constipation or diarrhea. Roentgen examination has shown atonicity of the stomach and changes in peristalsis. Some observers [15]

14. Harris, L. J., and Leong, P. C.: Vitamins in Human Nutrition: Excretion of Vitamin B_1 in Human Urine and Its Dependence on Dietary Intake, Lancet **1**: 886-894 (April 18) 1936.

15. Elsom, K. O.: Practical Aspects of Deficiency, M. Clin. North America **21**:1229-1244 (July) 1937.

have reported changes in the gastric hydrochloric acid, while others [4] have not found any departure from normal. There is evidence of impaired absorption of protein. Ulceration of the gastrointestinal tract, which, however, might be a cause of the deficiency rather than a result, has been reported,[17] as has swelling of the lymphoid patches in the ileum.[9] Narat and Losf [18] could not demonstrate stimulation of peristalsis by pure B_1 concentrates.

2. Neurologic symptoms are paresthesias, especially of the lower extremities, and impairment, and later loss, of vibratory sense in the toes. Polyneuritis has been observed, especially in alcoholism but also in pregnancy, diabetes and states of malnutrition.[4]

The primary neurologic lesion is apparently functional and due to faulty combustion of carbohydrates, resulting in a toxic accumulation of pyruvic acid. The microscopically demonstrated degeneration of the myelin sheaths is a secondary change.[19]

3. Cardiovascular symptoms are variable and not striking in moderate states of deficiency according to Elsom,[15] who reported dyspnea and persistent tachycardia. Others [20] reported vague reflex irritability, with bradycardia, asystole or syncope. No electrocardiographic changes were noted in one report,[15] while another [11] stated that changes in the T wave in 35 of 38 cases disappeared with B_1 therapy. The same observers reported dilatation of the peripheral arterioles with vasomotor collapse or vasoconstriction. Hypotension and failure of the right [15] or left [11] side of the heart have been seen in severe states. (The discrepancies in this account are apparently due to deficiencies of vitamin B of widely varying severity.)

4. Hematologic symptoms include macrocytic anemia similar to the pernicious form, which has been demonstrated in pregnant women. Ecchymoses of the lower extremity without trauma have been noted.

5. Edema is a prominent symptom and is present without sufficient renal or cardiac damage to explain it.

Other effects of deprivation of B_1 are said to be susceptibility to fatigue,[15] mental hebetude,[9] retardation of growth and development,[4] failure of lactation,[4] retinal hemorrhages, optic neuritis [21] and perhaps diseases of the ocular muscles.[11]

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16. Footnote deleted.

17. Mackie, T. T.: Ulcerative Colitis: Factor of Deficiency States, J. A. M. A. **104**:175-178 (Jan. 19) 1935.

18. Narat, J. K., and Losf, J. A.: Effects of Vitamin B_1 Concentrate (on Peristalsis and Blood Pressure), Arch. Int. Med. **60**:449-453 (Sept.) 1937.

19. (*a*) Booth and Hansen.[4] (*b*) Addinall, C. R.: The Story of Vitamin B_1, Rahway, N. J., Merck & Co., Inc., 1937.

20. Cited by Wilder and Wilbur.[11]

21. Booth and Hansen.[4] Wilder and Wilbur.[11]

Vitamin B_1 is said to be helpful in the treatment of herpes zoster ophthalmicus, neurosis of the sphenopalatine ganglion,[22] and certain forms of neuritis.[23]

Riboflavin.—Laboratory animals deprived of vitamin G (B_2) show retarded growth and loss of weight. Symptoms resembling scurvy and formation of cataracts have been reported in rats.[4] Dogs on certain diets deficient in vitamin G (B_2) showed marked demyelination of the peripheral nerves, the anterior and posterior spinal nerves and the posterior column.[6a]

In man the symptoms of riboflavin deficiency are not known.[4] It is presumed to be a dietary essential, and comparisons with deficiency states in laboratory animals have been made, but the results are largely conjectural.

Considering the whole so-called vitamin G (or B_2) complex (riboflavin, vitamin B_6 and the P-P factor, which has since been identified as nicotinic acid), Booth and Hansen [4] gave the following symptoms of deficiency in man:

1. Pellagra—due probably to avitaminosis P. P. . . .

Brown, scaly, symmetrical dermatitis in exposed areas, glossitis, soreness of mouth, indigestion, diarrhea, and disturbances of the nervous system—at times leading to dementia.

2. Acrodynia—believed by some to be caused by the lack of one or more of the factors in vitamin B complex [here including riboflavin]. Because of its cutaneous manifestations, acrodynia is often mentioned in connection with vitamin B_2 complex.

Irritability, insomnia, appearance of misery, anorexia, acrocyanosis, itching and burning of hands and feet, desquamation of palms and soles, marked perspiration, photophobia, muscular hypotonicity, increased blood pressure, and loss of teeth.

Relation with Endocrine Glands.—There are many suggestions of a relation between vitamin B and the endocrine glands. Booth and Hansen [4] stated that it is definitely linked with hormones. It may be related to the pituitary body and the adrenal cortex.[24] It is one of the essential elements of the thyroadrenopituitary group.[22] It is related to the endocrine secretions in rats.[25] Meurlin [25b] in a recent article stated that large amounts of thyroxin counteract the good effects of vitamin B_1 in rats and that, conversely, large amounts of vitamin B_1, because of the increase in metabolism, serve to offset the effects of thyroxin. He

22. Koepcke, G. M.: Vitamins and Infections of Eye, Nose, Throat and Sinuses, Journal-Lancet **57**:460-462 (Oct.) 1937.

23. Stevenson, D.: Vitamin B_1 in the Treatment of Neuritis, Practitioner **140**: 301-306 (March) 1938.

24. Selfridge, G.: Chronic Progressive Deafness, Mid-Winter Clinical Course of the Research Study Club, Los Angeles, 1938, privately printed.

25. (*a*) Rose.[2] (*b*) Meurlin, J. R.: Vitamins and Hormones, J. Am. Dietet. A. **14**:397-411 (June-July) 1938.

stated also that thiamin is synergistic to insulin in certain persons with diabetes but antagonistic in others. It is antagonistic to the diabetogenic hormone of the anterior lobe of the pituitary body. Some member of the B group other than riboflavin or thiamin has the power to prevent adrenal hypertrophy, which usually follows severe muscular exercise in certain animals.

Rhinologic and Otologic Symptoms.—A syndrome of vitamin B deficiency (fraction not determined) with special reference to rhinology was presented by Cody [26] in 1932. He listed the following symptoms:

> The complaint is a slight but frequent post-nasal discharge. . . . Sneezing is occasional but not in bouts. . . . Young women are more often affected. . . . The nasal mucosa on anterior inspection is . . . normal, except over the middle turbinates. Posteriorly, the tips of the turbinates have a smooth, moist, creamy-white appearance. . . . Mucus is seen in various locations, but not regularly in any one place.

It is noteworthy that nowhere was deafness mentioned except in Selfridge's articles (with one inconclusive exception),[27] but it certainly should be included as one of the possible clinical manifestations of deficiency of vitamin B. Kagawa and Naito [28] made no mention of deafness or of the eighth nerve in their report of experimental studies of B_1 deficiency in man. Analyzing Selfridge's case histories one finds that the other symptoms in his 14 cases of high tone deafness which improved with various forms of B therapy were as follows: low basal metabolism rate, in 8 cases; tinnitus, in 4; fatigue, in 3, low blood pressure, in 3; gastrointestinal disturbances, in 3; edema of Cody, in 2; dizziness, in 2; aural discharge, in 2, and allergy, loss of weight and vomiting, in 1 each. Aside from the symptoms referable to the ear, the clinical picture is one of low basal metabolism rate, with the patient below par, possibly with low blood pressure and gastrointestinal difficulties.

Summary of Symptoms.—In general, then, taking into consideration all the reported clinical data, one may say that a vitamin B–deficient person may have symptoms referable to the nervous, vascular or gastrointestinal system, the endocrine glands or the organs of special sense, will not have any pathognomonic lesion or syndrome and will be below par and in real danger of being relegated to the clinical limbo of "neurasthenic" patients.

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26. Cody, C. C., Jr.: The Relation of Vitamins A, D, B and G to Otolaryngology, Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:289-305, 1932.

27. Shambaugh, G. E., Jr.: Chronic Progressive Deafness Including Otosclerosis and Diseases of the Inner Ear, Arch. Otolaryng. **26**:583-605 (Nov.) 1937.

28. Kagawa, S., and Naito, H.: Die experimentelle B_1-Avitaminose beim Menschen, Jap. J. M. Sc., VIII, Int. Med., Pediat. & Psychiat. **4**:128*-129* (Feb.) 1936.

Laboratory Methods.—A syndrome by which to identify the patient in need of vitamin B therapy not being found, a search was made for a laboratory procedure which could be used to distinguish B–deficient persons. Harris [29] established that a 140 pound (63.5 Kg.) man excreting less than 12 international units of vitamin B_1 per day in the urine and failing to increase his urinary output if given a dose of 500 international units is suffering from a deficiency. Peters [6a] also stated that the urinary output of thiamin is diminished in the presence of a deficiency. Harris and Leong [20] gave the daily urinary output of a normal person as 12 to 35 international units, with an average of 20, which equals 5 to 8 per cent of the intake. Meiklejohn [20] gave the normal blood value of man as 4 international units per hundred cubic centimeters.

Now these values are precise, but difficulty is experienced in attempting to find out if one's patient comes up to the norm.

Harris and Leong [14] determined the B_1 content of the urine by a method which involved examination of a twenty-four hour specimen, adjustment to p_H 5 (bromcresol green indicator), extraction twice with claret acid clay (a form of fuller's earth) and biologic assay of the extract by quantitative feeding to B_1–deficient rats in which specific bradycardia had been produced sufficient to cause a fall in the electrocardiographic reading from 500 to 370 or 390. The time required for the heart rate to return to normal was the quantitative index, which was then compared with the amount of B_1 given certain normal control rats.

Roscoe [30] used a biologic method with vitamin B_1–deficient rats with neuritis and vitamin G (B_2)–deficient rats showing lack of growth. Using urine concentrates she concluded there was no relation between the presence or absence of vitamin B fractions in the urine of normal controls and the adequacy of the dietary intake.

Helmer,[31] studying human urine, used deprived rats for biologic assay.

Peters [8] used the restoration of avitaminotic brain tissue in vitro.

Arakawa [32] developed a colorimetric test for the milk of lactating women.

Schultz, Atkin and Frey [3] used the catalytic action on alcoholic fermentation.

Johnson [33] worked on the isolation of pyruvic acid in B_1–deficient pigeons. The following summary shows how elaborate the technic is:

29. Harris, L. J.: A Programme for Nutrition Surveys, Lancet **1**:966-968 (April 25) 1936.

30. Roscoe, M. H.: B-Vitamins in Human Urine, Biochem. J. **30**:1053-1065 (June) 1936.

31. Helmer, O. M.: Vitamin B_1 and B_2 Content of Human Urine, Proc. Soc. Exper. Biol. & Med. **32**:1187-1188 (April) 1935.

32. Cited by Booth and Hansen.[4]

33. Johnson, R. E.: Isolation of Pyruvic Acid from Blood of Vitamin B_1-Deficient Pigeons, Biochem. J. **30**:31-32 (Jan.) 1936.

After three preliminary procedures the blood precipitates stand for a week with a 0.6 per cent solution of 2, 4-dinitrophenylhydrazine. Then follow multiple adjustments of the p_H and extraction with sodium phosphate, five washings with hydrochloric acid and one with distilled water. This is followed by distillation in vacuo. The residue then undergoes multiple extractions, precipitations, washings and crystallizations, including three more distillations in vacuo. A bit elaborate for routine practice!

Meiklejohn,[34] for estimation of thiamin in the blood, used a modification of Schopfer's test, which is based on the catalytic action of B_1 for the growth of certain strains of molds.

Prebluda and McCollum [35] have developed a specific, sensitive chemical reagent which produces a purple-red compound with thiamin, which, they stated, is suitable for qualitative and quantitative estimation, but I did not find a technic practicable for routine clinical use.[36]

Westenbrink and Goudsmit [37] have developed a chemical method of quantitative determination of thiamin (aneurin) in urine by the thiochrome reaction (chemical conversion of thiamin to thiachrome). The procedure is somewhat elaborate and has certain phases in which inaccuracies may occur, and the final colorimetric reading is galvanometric, the norm being ascertained by running several specimens of urine to which known amounts of thiamin have been added. This is a step in the right direction, as it does away with deficient and control laboratory animals, but for routine use a simpler and more fool-proof procedure is necessary.

I conclude, then, that, as Helmer [31] stated in 1935 and Elsom [15] in July 1937, no satisfactory clinical test has been devised.[38]

STANDARDS OF MEASUREMENT

The literature on vitamin B contains references to many different units based on the potency of a clay adsorbate to relieve the symptoms of deficient laboratory animals. Since thiamin has been isolated and synthesized, future measurements of B_1 will undoubtedly be made in weight of pure thiamin chloride crystals. However, at the present time

34. Meiklejohn, A. P.: Estimation of Vitamin B_1 in Blood by a Modification of Schopfer's Test, Biochem. J. **31**:1441-1451 (Sept.) 1937.

35. Prebluda, H. J., and McCollum, E. V.: Chemical Reagent for Detection and Estimation of Vitamin B_1, Science **84**:488 (Nov. 27) 1936.

36. Naiman, B.: A Reagent for Vitamin B_1, Science **85**:290 (March 19) 1937.

37. Westenbrink, H. G. K., and Goudsmit, J.: The Determination of Aneurin (Vitamin B_1) in Urine by the Thiochrome Reaction, Rec. d. trav. chim. d. Pays-Bas **56**:7, 1937.

38. Clark, G. W.: Personal communication to the author. Hunsberger, A., Jr.: Personal communication to the author. Weigand, C. G.: Personal communication to the author.

it is necessary to have some means of comparing quantities, or rather potencies, expressed in different standards. The following definition, by Addinal,[19b] supplemented from other sources, is only approximate but contains the mean results of various competent investigators:

The international unit, adopted in 1934 by the Conference on Vitamin Standardization, is based on the potency of a clay adsorbate of an aqueous extract of rice polishings as tested for promotion of growth in deficient rats and cure of retraction of the head in deficient pigeons. It is typical of the hazards under which biochemists have labored that the daily dose of this "standard" preparation necessary to cure a rat is 10 to 20 mg. and that to cure a 300 Gm. pigeon 10 to 30 mg. This is considerable spread for a standard unit.

One international unit is equivalent to the following values:

3.333	micrograms of thiamin chloride
0.5	Smith curative unit
2.0	Sherman-Chase units
1.0	Roscoe unit
20.0	Cowgill milligram equivalents

This is the usual standard for pharmaceutic houses in the United States, the basis being 1 Gm. of thiamin chloride equals 300,000 international units.

The following observers, however, gave values for 1 international unit as follows: Peters,[6a] 2 to 4 micrograms of thiamin chloride; Harris and Leong,[20] 2 micrograms; Ohdake and Yamagishi,[39] 1 microgram; Elvehjem,[12] 3 micrograms and Ammerman and Waterman,[39] using Williams' crystals, 5 micrograms. Rose [2] stated that 1 Sherman-Chase unit equals 2.5 micrograms of thiamin chloride.

Vitamin G (or B^2) is measured in Sherman-Bourquin units, which are biologic units based on the growth of rats. So far as I know, this unit has not been converted into weight of riboflavin crystals.

REQUIREMENTS IN MAN

Cowgill,[40] realizing that the thiamin requirement is related to caloric intake and body weight, evolved the following formula, which has been widely quoted: $\frac{\text{vitamin B milligram equivalent}}{\text{caloric needs}} = 0.0000284 \times W$, in which W = body weight in grams. (One milligram equivalent of $B_1 = 1/20$ international unit.) When resolved for a man weighing 70 Kg., on a diet of 3,000 calories, the result is 300 international units per day. This is accepted by Gutman.[9] Jansen [32] in 1934 placed the daily adult require-

39. Cited by Elvehjem.[12]

40. Cowgill, G. R.: The Vitamin B Requirements of Man, New Haven, Conn., Yale University Press, 1934.

ment at 200 international units; Vorhaus in 1935, at 4,000 Sherman units; Harris in 1936, at about 1 mg. of crystalline thiamin chloride, and the Council on Pharmacy and Chemistry of the American Medical Association the same year, at 50 international units for children and 200 for adults. In 1937 Wilder and Wilbur [11] gave the value as 10 to 20 mg. of thiamin chloride. Rose [2] gave it as 10 Sherman-Chase units per 100 calories per day for adults and the same ratio for children. Peters [6a] put the adult daily requirement at 250 to 500 international units, or 1 mg. of crystalline thiamin chloride. Elsom [15] in 1937 estimated the minimum human requirement at 1,000 Sherman units per day, with the optimum intake at five times as much. Borsook [41] concluded that the minimum vitamin B_1 required to avoid beriberi is 1 international unit per pound (0.5 Kg.) of body weight per day and the optimum for health 5 to 7 international units per pound (0.5 Kg.) per day. It is generally agreed that the desideratum for health is about five times the minimum required to prevent beriberi.[42]

By assuming the subject to be on a 3,000 calorie daily diet and converting the values given into milligrams of crystalline thiamin chloride, one arrives at the following estimated daily requirements:

	International Units		Mg. of Crystalline Thiamin Chloride
Peters, Cowgill, Gutman, Harris	300	=	1.0
Jansen, American Medical Association	200	=	0.66
Borsook (minimum)	150	=	0.5
Borsook (optimum)	750 to 1,000	=	2.5 to 3.5
	Sherman Units		
Vorhaus	4,000	=	6.6
Wilder and Wilbur			10.0 to 20
Rose	300	=	0.5
Elsom (minimum)	1,000	=	5.0
Elsom (optimum)	5,000	=	25.0

At the risk of criticism for a facetious remark I cannot refrain from pointing out that these values, translated into loaves of white bread, mean a spread in daily consumption per person of from one and one-half to seventy-five! However, the general consensus seems to place the daily requirement at 300 international units, or 1 mg. of thiamin chloride, and Booth and Hansen [4] and others have stated that there are no ill effects from ingestion of many times this amount. It is, however, repeatedly stated in the literature [43] that lack of vitamin B is the

41. Borsook, cited by Smith.[43b]

42. Elsom.[15] Cody.[26]

43. (*a*) Elsom.[15] (*b*) Smith, H. A.: Personal communication to the author. (*c*) Selfridge, G.: Chronic Progressive Deafness from a Nutritional Standpoint: Preliminary Report, Ann. Otol., Rhin. & Laryng. **46**:875-894 (Dec.) 1937. (*d*) Other writers, too numerous to cite.

most glaring fault of the modern dietary, not only because of insufficient food for the poorer classes but from an overly refined diet among all classes. Borsook [41] stated the belief that principally because of the polishing of grain the average American receives only slightly more than the daily minimum. Sure [44] has stated that one third of the population of the country is under par from the standpoint of nutrition.

Certain conditions are known to modify the requirement of vitamin B_1: It is increased by a high intake of carbohydrate and is spared by high ingestion of fat.[15] It varies with the species and with the individual.[15] It is greater [2] in the presence of infection, fever, diabetes,[45] hyperthyroidism, pregnancy, exercise, a high basal metabolic rate, lactation, an unusual gain in weight, elderliness in women, a postoperative state and any gastrointestinal condition causing poor absorption, such as colitis, diarrhea, pyloric stenosis or achlorhydria.[15] The content of the diet may be adequate but utilization by the subject insufficient. Not only is the total ingestion important, but the concentration of thiamin in the diet influences its utilization.[15]

In regard to the requirements of riboflavin, Copping and Roscoe [46] stated: "There are at present [August 1937] no data available for calculating human requirements of vitamin B_2 complex." It is related to the protein intake.[4] An adequate intake has been estimated tentatively by Rose [2] and Daniel and Munsell [47] at 600 Sherman-Bourquin units for adults and 400 for children up to 10 years of age. Rose believes the optimum to be about four times the minimum necessary.

NATURAL SOURCES

The usual method of arriving at an assumption of deficiency of vitamin B is analysis of the dietary intake. The following table lists the most potent sources of vitamins B_1 and G (B_2) and the content of the commoner foods, in the approximate order of richness. It is compiled from tables in Addinall's publication [19b] and Rose's book [2] and from tables in the publication of Daniel and Munsell.[47] In Addinall's publication the figures were given for international units of thiamin per ounce (28.3 Gm.). In Rose's table (which is much more extensive than the list here submitted) the values for B_1 were expressed in

44. National Institute of Nutrition: Personal communication to the author.

45. Wilder, R. M., and Wilbur, D. L.: Diseases of Metabolism and Nutrition: Review of Certain Recent Contributions, Arch. Int. Med. **57**:422-471 (Feb.) 1936.

46. Copping, A. M., and Roscoe, M. H.: The Water-Soluble B-Vitamins in Yeast, Flour and Bread, Biochem. J. **31**:1879-1902 (Oct.) 1937.

47. Daniel, E. P., and Munsell, H. E.: Vitamin Content of Foods: A Summary of the Chemistry of Vitamins, Units of Measurement, Quantitative Aspects in Human Nutrition and Occurrence in Foods, Miscellaneous Publication 275, United States Department of Agriculture, June 1937.

arbitrary shares, one of which equals 10 Sherman-Chase units. For the sake of comparison these values have been converted into international units per ounce. The Daniel and Munsell publication gave figures sometimes in international units and sometimes in Sherman-Chase units per hundred grams. These have been converted into international units per ounce. There is a note in this table stating that the figure for thiamin is usually an indication of the amount of B complex present. The figures for B_2 from the last two sources are in Sherman-Bourquin units per ounce. There is qualitative agreement in the literature [48] as to the most potent sources, but the table illustrates the quantitative discrepancies so often found. These are in part due to analyses by different persons. The two figures so frequently appearing in the column based on the work of Daniel and Munsell represent the maximum and minimum in a list of observations made on different specimens at different times in different laboratories. A cipher indicates that the vitamin is absent; a dash, that no figure was given. One plus and 2 plus are qualitative indicators, meaning poor sources and fair sources, respectively.

	Thiamin, International Units per Ounce			Vitamin G (B_2), Sherman-Bourquin Units per Ounce	
Food	Addinall [19b]	Rose [2]	Daniel and Munsell [47]	Rose [2]	Daniel and Munsell [47]
Yeast					
Brewers', dried	—	709	200 to 1,180	326	500 to 1,000
Bakers', dried	—	142	>166	426	295 to 557
Bakers', compressed	—	50	—	336	—
Molasses, cane	210	0	0	—	—
Wheat					
Germ	190	170	75 to 625	86	—
Whole	28	21.5	28 to 113	24	16 to 33
Bran	56	22.5	—	+	—
Flour	4	0.5	—	0	—
Bread	3	0	—	±	—
Buckwheat	56	—	37	—	—
Rice					
Brown	28	15.5	18 to 33	0	—
Brown, boiled	21	—	—	—	—
Brown, polished	2	—	7 to 20	0	—
Rye germ	28	15.5	250	—	—
Corn					
Whole	28	+	24	+	—
Canned	14	—	—	+	—
Barley	26	—	18	—	—
Pearled	4	—	—	—	—
Beans					
Kidney	50	35	41	+	Trace
Lima	28	115	17 to 25	90	35
Baked navy	14	—	—	—	—
Soya	32	70	23 to 128	—	300
Peas					
Dried	36	85	25	100	35 to 50
Raw	21	42	13 to 50	28	35 to 80
Canned	11	+	—	8	—

(Table continued on next page)

48. H. J. Heinz Company has furnished nutritional data and charts corroborating this statement.

Food	Thiamin, International Units per Ounce			Vitamin G (B_2), Sherman-Bourquin Units per Ounce	
	Addinall [19b]	Rose [2]	Daniel and Munsell [47]	Rose [2]	Daniel and Munsell [47]
Meats					
Liver	46	14 (mutton)	13	258	265 to 333
Kidney	—	12.5	13	226 (beef)	265 to 333
		(pig)	113	—	—
Pork, lean	52	45	60	28	35
Ham	30	++	—	2+	—
Ham, cooked	—	0	—	2+	—
Bacon	25	+	—	24	28
Beef					
Muscle	7	+	1 to 25	28	33 to 50
Heart	—	—	17	—	100
Dried beef	14	—	25 to 33	2+	133 to 166
Mutton	7	17	20	2+	—
Veal	7	+	—	44	33 to 50
Chicken	7	—	—	+	—
White	—	21.5	50	—	—
Dark	—	43	100	—	—
Egg, whole	8	8	13	40	33
Yolk	14	25	20 to 43	66	50 to 100
White	—	0	0	28	20 to 40
Nuts					
Almond	28	11.5	27	—	—
Filbert	56	—	—	—	—
Hazelnut	56	—	66	—	—
Pecan	28	14	27 to 60	—	—
Walnut	17	42.5	50		—
Peanut	56	31	37	58	—
Peanut butter	—	20	—	36	—
Vegetables					
Asparagus	20	++	—	2+	—
Parsnip	20	10.5	12	—	—
Turnip greens	16	4.5	8	86	100
Avocado	—	14	17	16	18
Artichoke	14	+	—	—	—
Leek	14	—	9	—	—
Cauliflower	6	8	36	18	20
Cabbage	6	7	7 to 11	14	20
Potato	6	5.5	4 to 13	8	5 to 34
Potato, sweet	5	10	4 to 11	10	10 to 12
Carrot, raw	6	7	3 to 33	14	10 to 16
Carrot, boiled	4	—	—	—	—
Pumpkin	5	+	—	—	—
Broccoli	—	5	5 to 11	40	10 to 133
Tomato	—	5	3 to 13	98	4
Tomato juice	4	4.5	2	6	5 to 10
Lettuce	—	5	4 to 30	—	8 to 33
Squash	5	—	3	—	1
String bean	5	4.5	3 to 7	8	8
Spinach	4	11	6 to 13	6	16 to 50
Beet	4	1.5	—	14	16
Onion	4	1.5	13	4	—
Celery	4	++	Trace	—	—
Turnip	3.5	4.5	5 to 13	12	5 to 16
Dairy products					
Cow's milk	5	1.5	2.7 to 8	10	12 to 33
Human milk	3	—	1 to 7	—	6 to 11
Buttermilk	3	1.5	—	10	—
Cream	6	—	—	—	—
Butter	11	0	—	0	—
Cheese	3	+	0 to 10	14	14 to 96
Fruits					
Prune, dried	20	10	30	74	88
Apple, dried	9	—	—	—	
Apple, fresh	3	3.5	1 to 13	6	3 to 7
Banana	5	15	2 to 16	10	8 to 16
Cantaloup	3	3	<4	—	—
Orange juice	5	12.5	11	10	6 to 13
Lemon juice		+	—	+	—
Peach, fresh	3	—	2.7	—	20
Peach, canned	3	—	—	—	—
Sea foods					
Fish	6	—	13	+	80
Oyster	11	21.5	—	2	—
Clam	1	—	—	0	—

Because of the yeast used in brewing I thought that beer might be an excellent source of vitamin B. Two well known breweries furnished the following figures per 12 ounce (340 Gm.) bottle:

	Vitamin B_1, International Units	Vitamin G (B_2), Sherman-Bourquin Units
Company 1 [49]	35	61
Company 2 [50]	12	60

This is only 1 to 3 international units of thiamin and 5 units of vitamin G (B_2) per ounce (28.3 Gm.), which could not be considered a rich source. Most of the vitamin remains in the sedimentary yeast, but that which remains in the beer is not destroyed by the pasteurization.[49] No figures were furnished in direct answer to a question concerning draught (unpasteurized) beer.

It will be noted that the best sources of thiamin, namely, yeast, whole grains, wheat germ, beans, peas, lean pork, liver, kidney, nuts, asparagus, parsnips, turnip greens, avocados, artichokes, egg yolk and dried prunes, are the richest also in riboflavin. Liver and kidney are especially rich in the latter while relatively poor in the former, except pig kidney, which is high in thiamin. Egg white contains no thiamin, is relatively rich in riboflavin but does not contain the P. P. (antipellagra) factor.[4] Fish muscle is rich in the latter but not in riboflavin.[4] B_4 is not abundant in foods and is closely associated with B_1, as the latter cannot be obtained free of B_4 activity.[4] The distribution in foods of the remaining fractions of the complex is not known, but Copping and Roscoe [46] stated that they found all yeasts good sources of the B complex.

Variations in the Vitamin Content of Food Stuffs.—The vitamin content of original food stuffs varies considerably with climate, soil and maturity of the plant, or diet and age of the animal. Some of the discrepancies in the table are undoubtedly due to different methods employed, and certainly some error is present from converting figures from one biologic standard to another. But study of the Daniel and Munsell report shows, for example, wide variations between different species of the same bean. The different portions of a broccoli plant had varying values, and plants grown in Arizona were especially rich. Lima beans after freezing showed only a trace of thiamin, and oysters after freezing had lost two thirds of their value. The thiamin content of milk varied with the species of cow and still more with the diet. Pasteurization lowered the content 25 per cent in some instances and left only a trace in others. The loss of vitamin B_1 in the storage of rice amounted to 75 per cent in four years and varied with the container, while dried and stored peas had the same vitamin G (B_2) content as

49. Siemers, G. F.: Personal communication to the author.
50. Shakman, J. G.: Personal communication to the author.

fresh ones.[47] Another author [2] stated that peas in drying lose 50 per cent of their vitamin. Hanning [51] found peas twice as potent in 1934 as in 1932. Rose and Phipard [52] found peas of different seasons varying by 100 per cent. They also found that peas and lima beans lost half their B and G content by maturity. Other deleterious procedures are the treating of wines, ciders and other juices with bentonite (which absorbs vitamins B_1, G [B_2] and B_6) and the use of alkalis, as in the curing of ripe olives and the preparation of hominy.[43b] The values for flour are given in the table, but it is noteworthy that patent flour contains no vitamin B.[53] The riboflavin in flour is reduced by storage and bleaching but not the other B vitamins.[46] The riboflavin content of peas and lima beans was not altered by freezing.[52]

Effects of Cooking.—The greatest loss of thiamin in cooking results from its water solubility and heat lability in neutral [7a] or alkaline mediums. The loss of vitamin G (B_2) and the other factors is not great.[2] In boiling vegetables there is a great loss of thiamin if the water is discarded.[54] Even with the water retained Rose and Phipard [55] gave a loss in cooking peas of 25 per cent in fifteen minutes, beans 40 per cent and turnip greens 10 per cent. The addition of soda causes a rapid loss of B_1.[56] The amount of exposure to the atmosphere also is important.[57] In acid mediums, however, thiamin is relatively heat stable [7a] at cooking temperatures. Apples, for instance, according to Rose have the same value raw, baked and in applesauce. Boiling of oatmeal results in a 15 per cent loss.[2]

In regard to losses in baking, a large exposed surface as in preparing corn flakes,[2] or intense heat, as in preparing griddle cakes, results in a serious loss. The moisture content is a factor, as when all moisture is removed in the baking the temperature of the food rises to that of the oven or griddle.[56a] Copping and Roscoe [46] have made extensive studies on yeast and bread. They stated that, considering the dry weight of bread, there is little variation from the original flours but that the use

51. Hanning, F.: Vitamin B in Canned, Strained Vegetables: Further Studies, J. Am. Dietet. A. **12**:231-236 (Sept.) 1936.

52. Rose, M. S., and Phipard, E. H. F.: Vitamin B and G Values of Peas and Lima Beans Under Various Conditions, J. Nutrition **14**:55-67 (July) 1937.

53. Copping and Roscoe.[46] Daniel and Munsell.[47]

54. Rose.[2] Garnett, W. P.: Personal communication to the author. Smith.[43b] Other writers, too numerous to cite.

55. Rose.[2] Rose and Phipard.[52]

56. (*a*) Itter, S.: Personal communication to the author. (*b*) How We Get Our Vitamins, Special Circular, Extension Service, Department of Agriculture, University of Wisconsin, June 1936. (*c*) Other writers, too numerous to cite.

57. Jones, D. B.: Personal communication to the author.

of baking powder completely destroys the vitamin B_1. Others gave a loss of 10 per cent to 25 per cent in baking bread.[43b] The amount of yeast involved does not alter the vitamin content.[46] The quantity of vitamin B_1 in whole wheat bread (300 international units per pound [0.45 Kg.]) is three times that in white bread, but the amounts of riboflavin and of the vitamin G (B_2,) complex are more nearly the same for the two kinds of bread. Processing bran for breakfast food results in a 50 per cent loss.[2] One biscuit company reported a high figure for graham crackers but stated that this is variable owing to changes in the p_H of the dough.[58]

The Department of Agriculture [57] reported that commercial canning by modern methods results in much less loss than cooking in open containers. One canning company [59] stated that the whole process of cooking, canning and sterilizing may result in a loss of one-third the original B_1 content but that there is only negligible loss of the other B factors. Their published tables contain only qualitative information. Hanning [51] gave the results of her analysis of B_1 content of canned vegetables in international units per ounce (28.3 Gm.), which compare with the values for raw foods in the earlier tables as follows:

Tomatoes	7 to 12	=	No change
Peas	8.2	=	60% to 84% loss
Carrots	3.0	=	50% loss
Green beans	1.0	=	60% to 80% loss
Beets	1.0	=	33% to 75% loss
Spinach	1.6	=	60% to 88% loss

Another canning company [60] showed variable results in their published analyses. Tomatoes have about the value of the raw vegetable, while a compound of whole milk, whole wheat with added wheat germ and soybeans, which should certainly show a high B_1 content, has a value of only about that of raw milk.

From these facts it becomes evident that, even with the aid of Cowgill's formula or an assumed arbitrary standard of adequacy, dietary histories recorded by Watson's chart or dietary diaries kept by the patient are rendered almost worthless in the estimation of the intake (not to mention absorption) of vitamin B by the variability of the content of the original food stuffs and the losses through storing, processing and cooking. Even with weighed diets of known caloric value, the problem becomes one to tax the guessing ability of a trained dietary expert and is far beyond the otologist in actual office practice. The clinical laboratory does not help, and the alleged clinical manifestations of mild deficiency of vitamin B are protean and vague. Therapeusis

58. Lankenau, J. C.: Personal communication to the author.
59. Harding, E. R.: Personal communication to the author.
60. Libby, McNeil & Libby.

must be based for the present on an assumption made by the practitioner of the art of medicine rather than on a diagnosis substantiated by scientific means.

NOTE.—Since this was written Selfridge [61] has shown that the greater part of the improvement in hearing is due to nicotinic acid, while only a small fraction of the benefit is derived from thiamin.

REPORT OF CASES

The following epitomized case histories present the author's somewhat inconclusive experience with vitamin B treatment for impaired hearing:

CASE 1.—Mrs. S. A. W., aged 77, reported for treatment on June 17, 1938, complaining of loss of hearing for eighteen months, with roaring tinnitus. Tuning fork tests of the right ear showed impaired hearing throughout the scale, more marked in the lower portion, with shortened bone conduction and a raised low tone limit. In the left ear there was impairment throughout the scale, more marked for upper tones, with a lowering of the high tone limit. The eustachian tubes were normal on both sides. A diagnosis of mixed deafness was made and vitamin B complex made from rice bran prescribed, the daily dose being about 1,000 international units of vitamin B_1 and 200 Sherman-Bourquin units of vitamin G (B_2), the other factors being present in the same proportions as found in the natural bran. At the end of three months the bone conduction in the right ear had increased from ten seconds to fifteen seconds (normal) for the C-2 fork. The C-4 fork was not as well perceived, but the hearing for the C-5 fork was slightly improved. In the left ear the bone conduction was increased from eight to thirteen seconds, the perception of the C-4 fork from ten to thirteen seconds and that of the C-5 fork from three to five seconds. Measured by the Galton whistle, the high tone limit in the left ear, which had been 20,000, improved to 25,000. The patient's family had noted the improvement in hearing.

CASE 2.—Mrs. M. M. S., aged 65, was first examined on Sept. 14, 1937, stating that she had lost the hearing entirely in the left ear at the age of 12. There had been no suppuration, mumps, poliomyelitis or other ascertainable cause. The hearing in the right ear also was somewhat impaired and had become worse at the time of the climacteric. The patient did not know if the symptoms had been aggravated by pregnancies. She had two brothers and a sister deaf in the left ear. There was no deafness on the maternal side. The father was somewhat deaf, the condition coming on at about 70 years of age. The paternal grandfather was deaf. Bárány masking of the right ear showed total deafness of the left ear. Tuning forks demonstrated moderate impairment of hearing in the right ear throughout the scale, with slightly shortened bone conduction. The eustachian tubes were somewhat stuffy, the patient recently having had a cold. After a few inflations they opened well and stayed open. Vitamin B complex made from rice bran was prescribed, and after three months the results of testing were about

61. Selfridge, G.: The Eighth Nerve in Relation to Thiamin Chloride and Nicotinic Acid: A Comparative Study, Ann. Otol., Rhin. & Laryng. **48**:419-432 (June) 1939.

the same, with the exception that the C-4 fork was heard for fifteen seconds instead of ten seconds. The patient thought there was a slight improvement. There was no change in the totally deaf ear.

Case 3.—Dr. D. B. W., aged 42, first noticed slight high-pitched tinnitus in both ears at the age of 38, with slight difficulty in hearing, especially in crowds. The patient had had considerable urticaria and hyperesthetic rhinitis, but there had been no allergic manifestations for several years. There was a questionable history of healed pulmonary tuberculosis. There were no foci of infection. Repeated audiograms over four years had shown a practically stationary loss, beginning at C-5 and increased at C-6. After he had taken vitamin B complex for three months in amounts similar to those prescribed in case 1, the right ear showed a further loss of 10 per cent for the frequency 4096, while the left ear showed an improvement of 15 per cent throughout the lower portions of the scale. At 4096 the hearing was unchanged, while at 8192 there had been an improvement from 70 to 85 per cent. The tinnitus was somewhat less marked.

Case 4.—Mr. C. H. K., aged 38, was first seen on Jan. 20, 1938, when he stated that the hearing in the right ear had been somewhat less acute than that in the left ear for several years, becoming somewhat more impaired three years before. There was no tinnitus, nor had there ever been any suppuration. One brother was not deaf, but the mother had begun to lose her hearing before she was 40 years of age and had to use an artificial aid. The father was not deaf. The hearing of other relatives was unknown. There was no sinusitis. The tonsils were out and the teeth sound, and there were no symptoms referable to the general health. Hearing was impaired at both ends of the scale, with increased bone conduction. The eustachian tubes were entirely normal. A diagnosis of otosclerosis was made. The patient had a series of twelve injections of vitamin B_1, receiving 6.66 mg. of synthetic thiamin (2,000 international units) daily, and thereafter took vitamin B complex by mouth for two months, receiving a daily dose similar to that given in case 1. At the end of two months there was no change whatever in the the hearing.

Case 5.—Miss B. D. X. had been coming to my office for treatment for many years. In 1922 she had had encephalitis and had not been in robust health since that time. In 1929, at the age of 42, an artificial menopause was produced by roentgen rays. Subsequent to this transient attacks of light headedness developed, which eventually became attacks of actual loss of consciousness, in one of which she fell and injured herself. Deafness was first noticed in 1934, at which time a moderate impairment of perception for low tones was noted, with increased bone conduction. She had recurrent attacks of nasal allergy, during which the eustachian tubes closed. They were always restored to normal by inflations with a catheter and remained so between allergic attacks. The following year attacks of vertigo developed, precipitated by sudden movement and sometimes lasting several hours. Thorough examination for foci of infection revealed none. Fractional cold caloric tests elicited normal responses. There was no evidence of nerve deafness at that time. The attacks eventually subsided under treatment with large doses of ammonium chloride and have not recurred. The patient had been showing progressive shortening of bone conduction on the left side, and in January 1937, at the age of 50, she had a slight loss of perception of C-5 in the right ear and slightly shortened bone conduction in the left ear, with impairment of hearing for C-4 and C-5. The tone limits were normal. At that time there was no catarrh of the eustachian tube. Vitamin B_1 was prescribed, 2,000 units to be administered paren-

terally daily for one month, after which vitamin B complex was given by mouth in doses similar to those in case 1 for three months, at the end of which time hearing tests showed no change whatever.

CASE 6.—Miss B. T., aged 51, had had progressive deafness all her life, which had at first been diagnosed as conductive, but later was thought by two different otologists to be otosclerotic. She had been using an artificial aid for several years. Unfortunately, there was no more recent record of her hearing than two years prior to her taking large doses of vitamin B complex over a period of three months. However, at the end of that time hearing tests, compared with those of two years before, showed further slight impairment of hearing for C-2 by air and bone conduction, while perception of C-4 was slightly improved in both ears and that of C-5 unchanged.

CASE 7.—Mr. A. H. M., aged 65, was seen in February 1938, complaining of buzzing and deafness in the left ear for ten days, which he attributed to an occasional explosive sound over the telephone. There was moderate impairment at both ends of the scale, but in the Weber test the sound was referred to the good ear and the bone conduction markedly shortened in the worse ear, leading to a diagnosis of nerve deafness. The eustachian tubes were normal, and there were no foci of infection. After administration of vitamin B complex by mouth as in the previous cases for these months, hearing tests showed no change whatever. Twenty-five days later splenectomy was performed for idiopathic thrombopenic hemorrhagic purpura, and the patient did not recover. The systemic disorder might, of course, have been a factor in the nerve deafness of apparently acute onset.

Similar treatment has been prescribed in other cases, but it has been impossible to obtain follow-up reports because the patients live at a distance.

SUMMARY

The indexed literature of vitamin B has been reviewed up to January 1938, embracing a discussion of the chemical nature and physiologic effects of the various fractions, the clinical symptoms of deficiency of vitamin B, the various units employed, the natural sources and the human requirements.

It has been impossible to formulate a definite clinical syndrome for mild B deficiency.

A review of laboratory procedures failed to disclose one applicable to ordinary clinical practice.

Variations in the content of foods due to various factors render an estimation of the intake in the diet extremely inaccurate.

A review of my cases of deafness treated by vitamin B suggests that there is a possibility of helping some persons by this means but that at the present time there are no means of selecting the patients who may be improved.

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RECONSTRUCTION AFTER RADICAL OPERATION FOR OSTEOMYELITIS OF THE FRONTAL BONE

EXPERIENCE IN EIGHTEEN CASES

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Since 1931, at the suggestion of Dr. H. P. Mosher, a radical operation for osteomyelitis of the frontal bone has been used routinely at the Massachusetts Eye and Ear Infirmary. Because of the poor prognosis with conservative treatment, radical resection of the frontal bone has been adopted as the operation of choice at other hospitals as well.

The adoption of this radical operation has greatly diminished the mortality from the disease. For instance, statistics show in 1928 and 1930 a total of 6 cases, with a mortality of 83.3 per cent—only 1 patient out of 6 surviving; during the years 1936, 1937 and 1938,[1] 8 patients were treated without a mortality.

The new operation, however, has created another problem, namely that of the deformity. It is unnecessary to enlarge on its psychologic, social and economic consequences for the patient.

Eighteen patients have been referred to the plastic service of the Massachusetts Eye and Ear Infirmary for repair of the deformity. Eleven patients were from the clinic; 7 were private patients. Eleven were males; 7 were females. The ages ranged between 8 and 44 years. Fourteen patients, or approximately 75 per cent, were under the age of 25. Eleven patients, or approximately 60 per cent, were under the age of 21.

Just as is radical resection of the frontal bone, plastic repair of the consequent deformity is now a standardized procedure at the Massachusetts Eye and Ear Infirmary.

Being concerned in this paper essentially with the plastic repair, we do not wish to discuss osteomyelitis of the frontal bone. On that

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From the plastic clinic of the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary.

Read before the Section on Laryngology, Otology and Rhinology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 18, 1939.

1. Until Sept. 1, 1938.

subject we refer the reader to other publications.[2] Ten of the cases in this series have been reported in two previous papers, by Drs. Mosher and Judd [3] and by Dr. Mosher.[4]

OPERATION FOR OSTEOMYELITIS

Briefly summarized, the manner of treating osteomyelitis of the frontal bone in this hospital is as follows: The object of the operation being the complete eradication of the diseased focus and the prevention of recurrence, the frontal bone, after incision of the skin and periosteum and exposure, is removed en masse with the Hudson drill. Trephine holes are made in healthy bone away from the diseased area, and are joined with the Gigli saw (fig. 1 *A*). The anterior and posterior walls of the frontal sinuses are removed with rongeurs. After the operation there is a large defect in the bone in the frontal area, limited by the edges of the surrounding bone, and an area of dura of corresponding size is exposed. The wound is carefully packed so that the skin flaps, along with the periosteum, are kept elevated and turned away from the edges of the defect in the bone to permit their regular inspection for recurrence or formation of sequestrums (fig. 1*B*). Until these possibilities are eliminated, along with that of intracranial complications, there is no question of plastic closure.

PROBLEM OF PLASTIC CLOSURE

The patient presents himself with a large gaping wound in the forehead (fig. 2). A large area of dura lies uncovered and unprotected by bone. The problem from a plastic point of view is twofold: first, that of closing the defect in the soft tissue by an approximation in proper position of the skin flaps; second, that of replacing the absent bone.

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2. (*a*) Furstenberg, A. C.: Osteomyelitis of the Skull: The Osteogenetic Processes in the Repair of Cranial Defects, Ann. Otol., Rhin. & Laryng. **40**:996 (Dec.) 1931; The Pathology of the Spread of Osteomyelitis of the Skull: Its Relation to Brain Abscess, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:423, 1933. (*b*) Adson, A. W.: Surgical Treatment of Osteomyelitis of the Skull, Proc. Staff Meet., Mayo Clin. **8**:169 (March 15) 1933. (*c*) Behrens, H. C.: Osteomyelitis of the Skull of Otitic and Paranasal Sinus Origin, Arch. Otolaryng. **25**: 272 (March) 1937. (*d*) Adson, A. W., and Hempstead, B. E.: Osteomyelitis of the Frontal Bone Resulting from Extension of Suppuration of the Frontal Sinus: Surgical Treatment, ibid. **25**:363 (April) 1937.

3. Mosher, H. P., and Judd, D. K.: An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis, Laryngoscope **43**:153 (March) 1933.

4. Mosher, H. P.: Osteomyelitis of the Frontal Bone: Notes on Three Cases, J. A. M. A. **107**:942 (Sept. 19) 1936.

TYPE OF INCISION

The type of incision which is made at the time of the initial operation for the removal of the osteomyelitic frontal bone necessarily has a bearing on the secondary plastic operation. The primary object of the cutaneous incision is the proper exposure of the frontal bone. How-

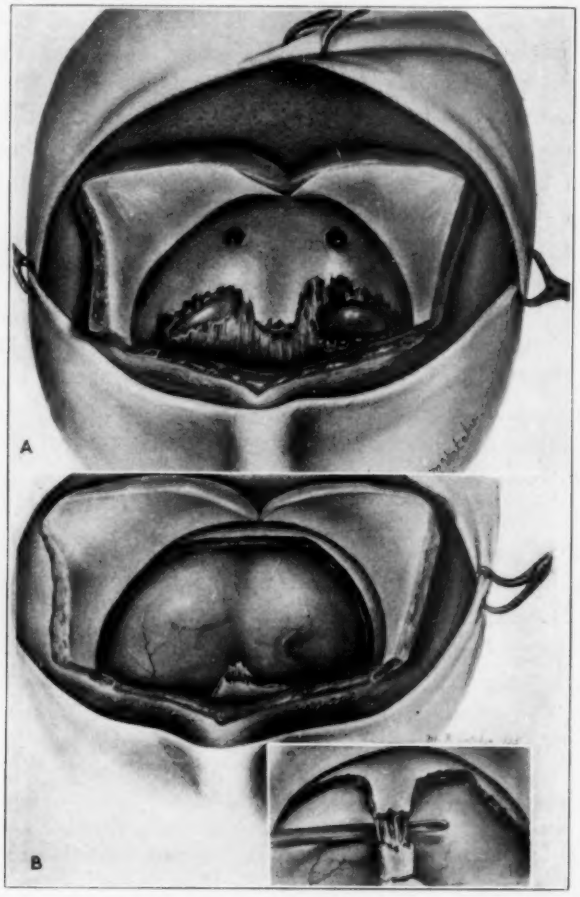

Fig. 1.—*A*, diseased bone exposed and drill holes made at a distance from the osteomyelitis. *B*, frontal bone resected; a portion of the anterior wall of the left frontal sinus still remaining, to be removed along with the posterior wall.

ever, in the process of repair objections were found to certain types of incision. Modifications of the incisions were proposed, which did not handicap the operator but greatly facilitated subsequent repair. As much as possible, an effort has been made to bear this consideration in mind and to use the most favorable type of incision.

The inverted U incision (fig. 3 *A*), ideal cosmetically, because sparing an incision through the lower part of the forehead and the eyebrows, proved to give insufficient exposure of the lower portion of the frontal bone and the frontal sinuses and to be unfavorable to surgical drainage.

The straight U incision (fig. 3 *B*) was attended with considerable difficulty in keeping the flap elevated and sufficiently retracted to afford an inspection of the upper edge of the defect in the frontal bone, its natural tendency being to fall down over the wound. In the course of the plastic repair difficulties were encountered in dissecting away from the dura and in bringing down the curled-up flap. From a plastic point

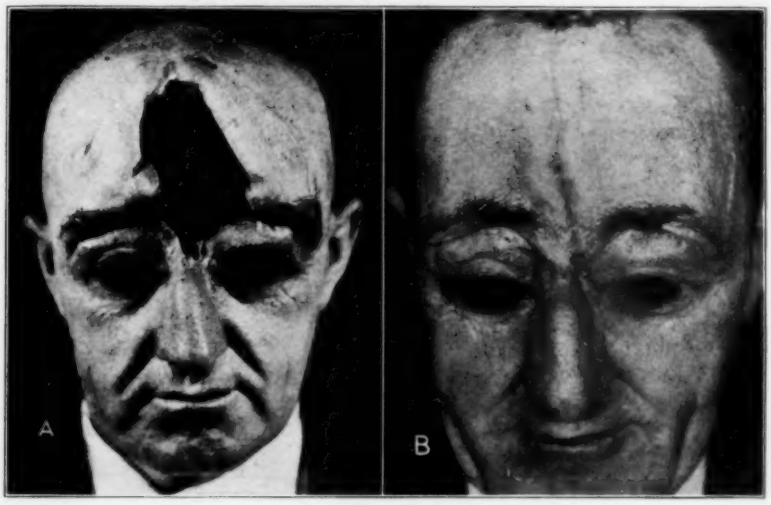

Fig. 2.—*A*, patient with large, gaping wound in the forehead, following the removal of a large section of the frontal bone. *B*, patient after closure of the wound, fourteen days after operation.

of view the straight U incision appeared to present only disadvantages over the inverted T incision.

The latter consists of a vertical branch extending upward in the midline of the forehead, above the area of osteomyelitis indicated by the edema, usually above the hairline, and a horizontal branch passing through, above or below the eyebrows.

At first the horizontal incision was passed through the eyebrows (fig. 3 *C*), with the idea of dissimulating part of the scar. It was found that when the skin flaps were brought together the eyebrows were considerably distorted, because one half was carried up with the reflected skin flap while the other half remained in its proper position. When the

reflected flap was secondarily brought back into position, the eyebrows could not always be exactly fitted. Often a wide hairless scar separated the upper from the lower half of the eyebrows. This distortion was marked enough to necessitate special subsequent operative correction.

It was therefore decided to avoid incision through the eyebrows and to adopt an incision either above or below them.

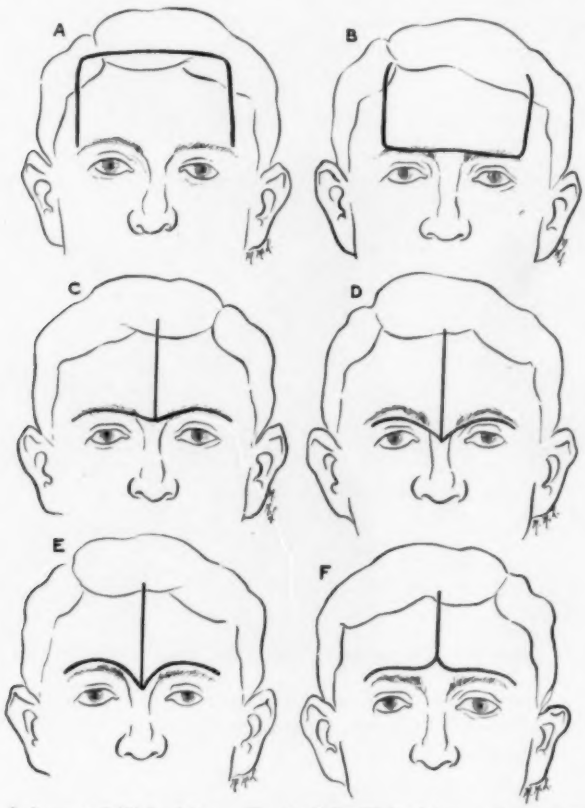

Fig. 3.—*A*, inverted U incision. *B*, straight U incision. *C*, inverted T incision passing through the eyebrows. *D*, inverted T incision below the eyebrows. *E*, inverted T incision carried just above the eyebrows. *F*, inverted T incision above the eyebrows, with rounded angles, the most desirable type of incision for exposure of the frontal bone.

The incision below the eyebrows (fig. 3 *D*), while giving a slightly better exposure of the front face and floor of the frontal sinuses, presented the disadvantage over the incision above them that they were completely carried away from their normal position by the skin flaps.

The incision carried just above the hairline of the eyebrows appeared, therefore, the most favorable (fig. 3 *E*).

It was noticed, however, that at the point of junction between the horizontal and vertical branches localized sloughing occurred rather frequently when the flaps were sutured together, apparently because this is a meeting point of angles of tissues of diminished blood supply. It was found advisable to round off the angles. The most suitable incision has appeared to be that illustrated in figure 3 *F*.

POSTOPERATIVE CARE AND PERIOD OF OBSERVATION

When one has performed the resection of the frontal bone, using the most favorable cutaneous incision, the question arises as to how soon the first stage of plastic repair, or closure of the soft tissues, may be performed. After the radical removal of the frontal bone, the wound is kept clean with sterile solution; if secondary infection of the wound

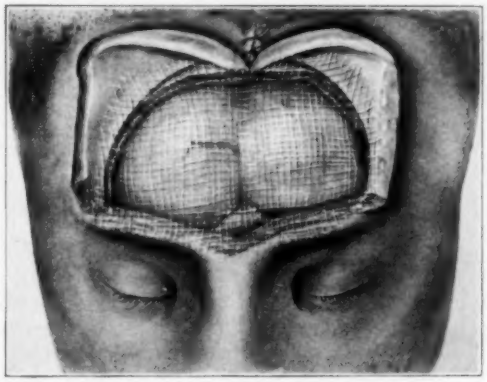

Fig. 4.—Coarse paraffin mesh protecting the dura from the dressing in the early stages.

should set in antiseptics such as dilute solution of sodium hypochlorite may be used; bacteriophagic dressings and urea solution have occasionally been used. A nonadherent perforated tissue, such as "silkaloid," protects the dura from the dressing in the early stages (fig. 4). During this period attention is focused on watching the edges of the defect in the bone for recurrence of the osteomyelitis and for sequestrums by stripping back the flaps along with the periosteum and on detecting possible intracranial complications.

Experience gained in this series of eighteen cases has shown that the plastic closure should not be performed until all suppuration has ceased and the wound has become filled with healthy granulations. An excellent index of the time to operate is the presence of a thin layer of newly formed epithelium covering most of these granulations. This process usually takes two to three months. If the wound is closed at

an earlier period it appears impossible to be sure that all suppuration has ceased. Pus may be pocketed off in small areas under one or both flaps. In 1 case, closure was performed forty-two days after the removal of the frontal bone; an abscess of the brain developed, which was found two months later and drained. The patient subsequently died of meningitis; although this result cannot be attributed directly to the plastic closure, the fact remains that closure was performed too early.

It was noticed also that in practically all the cases definite marginal sequestration of bone occurred from thirty to sixty days after the operation. In 1 of the cases, after repeated cultures showing absence of active infection, closure of the wound was performed, fifty-two days after the operation. There followed troublesome months, during which subcutaneous abscesses were incised and sequestrums were removed.

In conclusion, therefore, it appears that haste should not be taken in performing the plastic closure after the resection of the frontal bone for osteomyelitis. Delay of at least three months is advisable.

FIRST STAGE OF PLASTIC REPAIR

The first stage of the plastic repair, closure of the defect in the soft tissue, consists in bringing down the skin flaps from their position curled up over the edges of the bone to their original site.

The operation has been performed both under a general anesthetic, usually ether, and under local anesthesia induced by infiltration of a 2 per cent solution of procaine hydrochloride with a 1:50,000 solution of epinephrine; it is carried out in the following successive stages:

1. The skin flaps are dissected away from the dura and the surrounding bone (fig. 5 *A* and *B*). The flaps are found curled up, apparently because of the pull of the fibroblasts and elastic fibers of the subepithelial tissue.

2. The thin layer of new epithelium is shaved off with a knife (fig. 5 *C*).

3. To break the pull of the deep layer and to permit liberation of the flap a series of crisscross incisions is made with the knife through the subepithelial tissue (fig. 5 *D*).

4. The flaps are then drawn down into their original position and sutured with the finest silk sutures after careful trimming of the edges of the skin to insure perfect approximation (fig. 5 *E*).

Careful hemostasis is essential. Finally a mildly compressive dressing is applied. It is not unusual to meet small pus pockets or necrotic bone in the process of dissecting the flaps. Careful placing of rubber drains and the removal of the necrotic bone usually prevent complications in such cases.

After this plastic closure the appearance of the patient is considerably improved (fig. 2*B*). Instead of an ugly, irregular wound, there is a smooth forehead with a continuous covering of skin. There is still, however, noticeable deformity due to the absence of bone. From the front of the face, a depression can be seen. Under the skin, pulsations of the brain can be perceived, which are distinctly felt by the patient. In profile, flattening of the frontal region gives the patient a

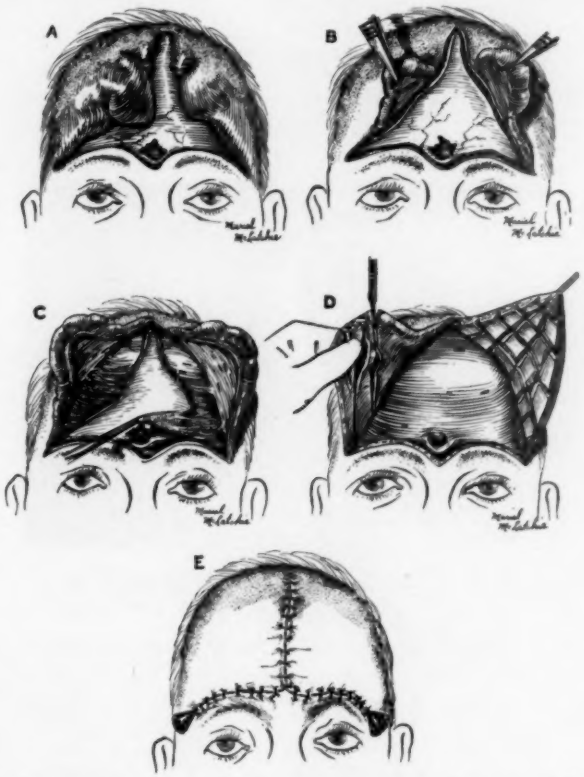

Fig. 5.—*A*, wound of three months after resection of the frontal bone. *B*, skin flaps dissected back, exposing the surrounding bone; superficial epithelium incised. *C*, epithelium dissected over the layer of granulations covering the dura. *D*, incisions carried through subepithelial tissue to permit extension of the skin flaps. *E*, skin flaps sutured into position, with drains in place.

peculiar appearance, "birdlike" in extreme examples (fig. 6). Moreover, the patient is usually worried about the presence of the frontal lobes under the skin. He becomes haunted by the thought of possible injury and in most cases wears a protective headpiece over the defect in the bone.

The extent of the deformity is naturally directly related to the amount of frontal bone removed and to the height of the forehead. In a patient with a low forehead the deformity is not as noticeable, since the hair compensates greatly for the lack of fulness of the frontal region.

The defect in the frontal bone is irregularly quadrilateral and varies from 7 to 12 cm. in width and from 6 to 10 cm. in height. It is evident, therefore, that a second stage in plastic reconstruction is indicated, to replace the lost bone.

REGENERATION OF BONE

The question arises at this point how much regeneration of bone one can expect and how long one should wait for it to occur.

Fig. 6.—Extreme flatness of the forehead.

A revision of the roentgenograms made in the cases of this series was done by Dr. A. S. MacMillan. It appeared that there was regeneration of bone in 2 cases. The regeneration was complete and was observed about eighteen months after the removal of the frontal bone. Both patients were children, aged 8 and 9 years respectively (fig. 7). In none of the 16 other cases was any definite regeneration observed. The youngest patient in these 16 cases was 13. Roentgenograms were taken periodically in all the cases; some patients were followed for six years after the removal of the frontal bone.

Why should there have been regeneration in the cases of the 2 children and none in the other cases? According to Gray the skull grows rapidly from birth to the seventh year, by which time the foramen magnum and the petrous parts of the skull have attained their full size. Around puberty a second period of activity occurs; this results in an increase in all directions, but it is especially marked in the

frontal and facial regions, where it is associated with the development of the sinuses.

That bony regeneration is remarkably active in children is confirmed by the complete obliteration of the frontal defect in the 2 children. In another case, not included in this series, that of a boy aged 8 years, in which conservative treatment was used on account of the widespread extension of the osteomyelitis throughout the skull, regeneration of nearly the whole calvarium was observed.

In the other 16 cases of this series no regeneration of bone was found. This observation is generally confirmed by a review of the literature on the subject, and the accepted opinion that the flat bones of the skull do not regenerate appears to be substantiated.

That there are exceptions is evident. Furstenberg [2a] cited numerous examples and gave a whole series of cases in which bone was regener-

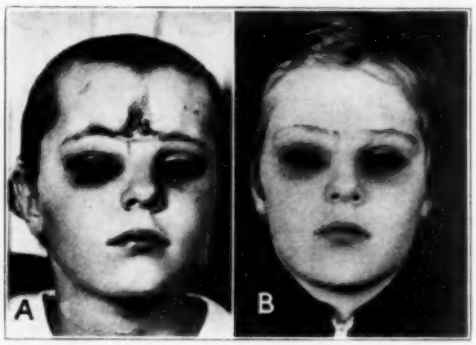

Fig. 7.—*A*, patient before plastic repair. *B*, patient after plastic closure and spontaneous regeneration of bone.

ated. If there is regeneration in certain cases and not in others, what is the determing factor?

If the periosteum is responsible for regeneration of bone it is given a poor chance to survive. After the resection of the frontal bone the skin flaps and the periosteum are reflected back to expose the edges of the remaining bone. Rapidly the natural tendency of the periosteum and of the subcutaneous tissue to shrink causes the skin flap to curl on itself. The periosteum tends to disappear under the flap and ceases to remain a distinct structure, lost as it is in an amalgam of newly formed granulations. Therefore, when the plastic closure is done and the skin flaps are brought down into their former position, the periosteum has ceased to exist as such and the periosteum of the surrounding bone is probably incapable of producing new bone, because for regular inspection of the edges of the bony defect it must be stripped up from the bone and this maneuver repeated regularly may produce an area

of devitalized bone, lacking a normal periosteum, which encourages sequestration, and inhibits bony regeneration.

If this procedure, however, were not carried out in the early stages and the wound were allowed to close it would be impossible to watch for progression or recurrence of the osteomyelitic process except by roentgen examination, which often reveals the presence of osteomyelitis only as late as ten days after the direct inspection of the bone would have revealed it (Mosher).

To explain regeneration of bone, Furstenberg expressed the opinion that the dura and even the adjacent connective tissue may become osteogenetic. In 2 recent cases, not included in this series, triangular flaps of normal periosteum were brought down from the upper frontal region to lie over the bony defect prior to suturing the skin flaps. In bringing down this periosteum it was turned over on itself, the side toward the normal bone above becoming the outer side. Regeneration has not occurred in these 2 cases after five months and after three months.

Judging from the experience gained in this series, it appears that it is impossible to rely on spontaneous regeneration of bone and that the bone must be replaced by operative measures. The second stage of the plastic reconstruction for the closure of the defect in the frontal bone will now be considered.

SECOND STAGE OF PLASTIC REPAIR

Since the World War numerous reports have been made of methods for replacing loss of bone in defects of the skull. A review of the literature showed that most of the methods proposed for the closure of such defects apply to much smaller ones than those following resection of the frontal bone. Most of the defects followed operative trephining or shrapnel or bullet wounds. For these the use of an osteoperiosteal graft removed from the outer table of the skull, as proposed by Mauclaire,[5] Müller and Koenig,[5] Jones,[6] Reed,[7] Frazier and Ingham,[8] Coleman[9] and Gurdjian,[10] appears ideal. For larger defects,

5. Cited by Juvara, E.: Procédé de cranioplastie: Reconstitution de la paroi cranienne par des greffes, minces lames osseuses découpées à la scie de la corticale interne du tibia, Rev. de chir., Paris **52**:401 (June) 1933.

6. Jones, R. W.: Repair of Skull Defects by New Pedicle Bone-Graft Operation, Brit. M. J. **1**:780 (May 6) 1933.

7. Reed, J. V.: Repair of Cranial Defects, Am. J. Surg. **18**:285 (Nov.) 1932.

8. Frazier, C. H., and Ingham, S. D.: Review of the Effects of Gunshot Wounds of the Head Based on the Observation of Two Hundred Cases at U. S. General Hospital No. 11, Cape May, N. J., Arch. Neurol. & Psychiat. **3**:17 (Jan.) 1920.

(Footnotes continued on next page)

various methods have been proposed. Plates made of silver or [11] celluloid [12] have been universally recognized as unsuccessful. Boiled bone [13] and bone removed from cadavers have been used. Naffziger [14] gave an account of the use of pathologic bone removed from the skull, sterilized and transplanted back to its former site with success. Gurdjian [10] used boiled bone removed from the skull of a cadaver after sterilizing it and observed its resorption within eighteen months. Pankratiev [15] described 4 successful transplants of sterilized bone from cadavers.

O'Connor [16] used large grafts of beef cartilage supplemented by small cartilaginous isographs to fill in frontal defects.

Cartilage removed from the ribs was used extensively during the war, particularly by Morestin.[17] While an excellent transplant material, cartilage has the inconvenience of having to be used in multiple pieces, as a single piece large enough cannot be obtained; moreover, it affords little protection, as it merely becomes encapsulated in fibrous tissue without cementing itself to the adjacent bone.[18] It was used extensively in some of the cases reported in this paper to fill in certain minor defects following the closure of the defect by the bone graft.

Osteoperiosteal grafts appear to be most practical, because they are easy to obtain and become joined to the adjacent bone to form a continuous protective bony covering. Grafts have been obtained from the scapula,[19] the ribs and the ileum, but the most satisfactory source is the tibia. Easily accessible, the tibia yields the large flat grafts that are needed to repair large defects.

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9. Coleman, C. C.: Repair of Cranial Defects by Autogenous Cranial Transplants, Surg., Gynec. & Obst. **31**:40 (July) 1920.

10. Gurdjian, E. S.: Management of Depressed Fractures of the Skull and Old Skull Defects, Ann. Surg. **102**:89 (July) 1935.

11. Elsberg, C.: Plate for Defects of the Skull, Ann. Surg. **47**:795, 1908.

12. Mitchell, A. B.: Repair of Injuries to the Skull by Perforated Plates, Brit. J. Surg. **5**:40 (July) 1917.

13. Babcock, W. W.: Soup Bone Implant for the Correction of Defects of the Skull and Face, J. A. M. A. **69**:352 (Aug. 4) 1917.

14. Naffziger, H. C.: Restoration of Defects in the Skull, with Special Reference to the Management of Intrinsic Tumors of the Skull and Certain Types of Localized Osteomyelitis, Ann. Surg. **104**:321 (Sept.) 1936.

15. Pankratiev, B. E.: Deaf Bone Grafts to Repair Skull Defects, Ann. Surg. **97**:321 (March) 1933.

16. O'Connor, G. B.: Contour Reconstruction After External Frontal Sinus Operation, Ann. Otol., Rhin. & Laryng. **47**:138 (March) 1938.

17. Morestin, cited by Woodroffe, H. L. W.: The Reparation of Cranial Defects by Means of Cartilaginous Grafts, Brit. J. Surg. **5**:42 (July) 1917.

18. Primrose, A.: Cranioplasty: The Value of a Graft of Bone, Cartilage or Fascia in Closure of Cranial Defects Caused by Wounds of War, Ann. Surg. **70**:1 (July) 1919.

19. Jones, B. L.: Bone Transplantation from Scapula, Ann. Surg. **66**:160 (Aug.) 1917.

Delangenière [20] reported 104 cases of successful transplantation by this method. He put the periosteal surface against the dura. If necessary he used two layers of graft with the rough bony surfaces facing each other.

Albee,[5] to obtain the necessary curvature of the graft, made a series of parallel lines on its under surface with the electric saw, rendering it possible to bend it to the desired shape.

Juvara [21] laid the graft over the dura, its internal or rough surface inward, having previously smoothed it with a large file. He used a special forceps to bend the graft to the suitable shape.

Similar methods, slightly modified, were reported by Tillier,[22] Brenizer [23] and Rhodes.[24]

In this group of 18 cases, bone grafting was carried out in 4. In 2 cases of children, the patient presented complete regeneration. In the other 12, either the patient did not care to undergo an additional operation, or sufficient time has not elapsed at the time of writing for the operation to be performed.

In all the cases sufficient time was allowed to permit bony regeneration, the period between the closure of the soft tissues (first stage) and the bone grafting (second stage) having been from twelve to eighteen months.

The operation for transplanting bone to the frontal defect has been performed under general anesthesia, under local anesthesia, and under a combination of both methods (avertin with amylene hydrate and a local anesthetic).

1. Incisions are made through the scars in the frontal area, and the flaps are dissected away from the dura until the edges of the surrounding bone are encountered.

2. The periosteum covering the surrounding bone is elevated over about 2 cm., and the bony edges are freshened with a rongeur.

3. Careful hemostasis of the wound is then carried out.

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20. Delangenière, H., and Lewin, P.: A General Method of Repairing Loss of Bony Substance and of Reconstructing Bones by Osteoperiosteal Grafts Taken from the Tibia, Surg., Gynec. & Obst. **30**:441 (May) 1920.

21. Juvara, E.: Procédé de cranioplastie: Reconstitution de la paroi cranienne par des greffes, minces lames osseuses découpées à la scie de la corticale interne du tibia, Rev. de chir., Paris **52**:401 (June) 1933.

22. Tillier, R.: Cranioplasty for Large Loss of Frontal Bone, Bull. et mém. Soc. nat. de chir. **56**:1277 (Nov. 29) 1930.

23. Brenizer, A. G.: Total Bone Grafts into Skull Defects, Ann. Surg. **64**:516 (Nov.) 1916.

24. Rhodes, R. L.: Treatment of Cranial Defects by Bone Grafting, Surg., Gynec. & Obst. **19**:546, 1914.

4. An incision is made medially to the crest of the tibia, exposing its inner aspect. The length of the incision is determined by the length of the graft to be removed and is generally from 10 to 15 cm. After a good exposure of the bone covered by its periosteum a wide, flat and rather thick (about 0.5 cm.) osteoperiosteal graft is removed with a wide chisel. In certain cases one wide graft is sufficient, but generally two grafts are necessary.

5. Each graft is grasped between compresses and fractured into the proper curvature to reproduce the natural contour of the forehead.

6. The grafts are then introduced, one in the lower and the other in the upper portion of the defect. They are placed with the periosteal surface outward, the ends in contact with the edges of the surrounding bone; the periosteum of the graft is sutured to the periosteum of the adjacent bone.

7. The skin flaps are sutured together with fine silk, and a mildly compressive and immobilizing dressing is applied.

A systematic check-up by roentgenograms, carried out over years at the Massachusetts Eye and Ear Infirmary, has shown that the grafts have been successful.

The appearance of the patient approaches that of a normal person. The contour of the forehead is satisfactory, and the grafts are firm, solid and resistant (fig. 8).

The reasons for not transplanting bone at the initial operation are as follows:

1. The occurrence of intracranial complications cannot be excluded even at this stage; therefore, it is felt that it is safer to repair the cutaneous defect first.

2. As most of the patients have large frontal defects, the operative trauma is considerable. Moreover, because of the possibility of infection, it is advisable to take no chances of failure of the bone graft. With small defects, when the entire wound is covered by epithelium, it may be safe to transplant bone at the initial operation for the repair of the defect. This was successfully done in 2 cases.

3. Usually an opening at the nasofrontal duct communicating with the nasal cavity is present at the first operation.

Transplantation of bone improves the appearance of the patient and contributes added protection. However, it was found that in order to gain further cosmetic improvement in some cases it has been necessary to add pieces of cartilage over the eyebrows and frontal eminence. In 1 case a large piece of dermal graft was inserted over the entire

forehead four months after the operation for bone graft, with a rather pleasing effect on the contour of the forehead.

Cartilage has been used thus only as a secondary method, for the restoration of the contour and a completion of details impossible to

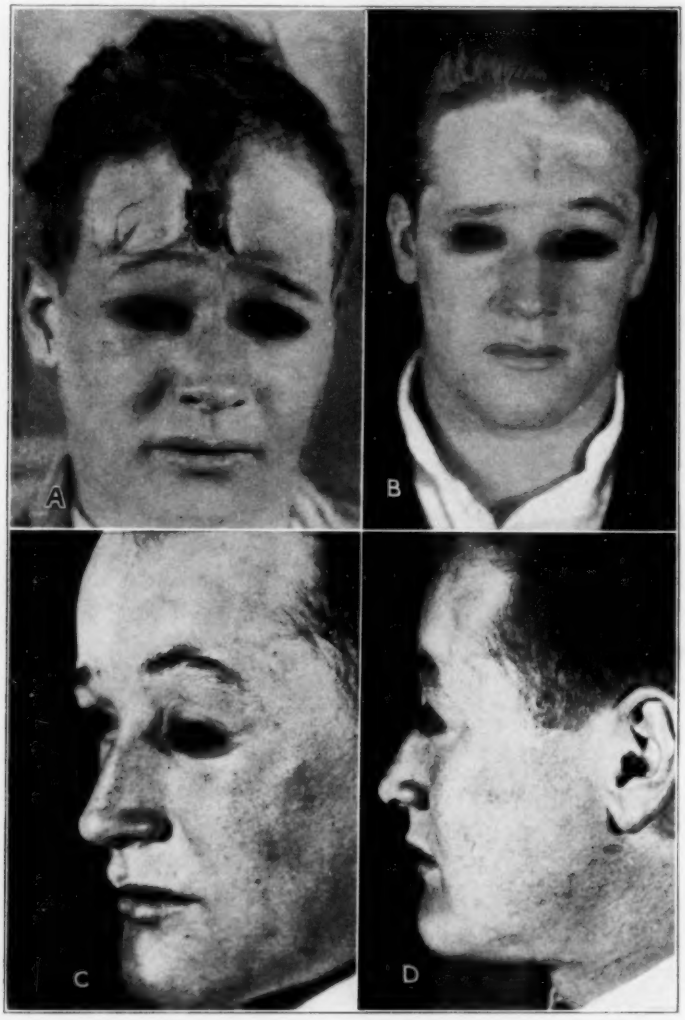

Fig. 8.—*A*, patient six months after resection of the frontal bone. *B*, patient after plastic closure of the skin flaps; depression due to absence of bone. *C* and *D*, patient after bone grafting; restoration of the normal contour of the forehead.

achieve with large bone grafts. For this, grafts are removed from the cartilage at the junction of the seventh, eighth and ninth ribs, shaped and buried under the skin in desired locations.

COMPLICATIONS

Complications following the plastic repair were due to too early closure and have been previously reported. One patient died of abscess of the brain and meningitis. Although the complication cannot be attributed directly to this, the fact remains, as already mentioned, that closure was performed too early. In another case the elimination of sequestrums continued for months after an early closure of the flaps.

The possibility of convulsions following the plastic repair, due to adhesions between the dura and the skin or the grafted bone, has been considered. Epilepsy as a sequela of osteomyelitis was observed in 3 of the 18 cases reported. In all 3 cases the convulsions had occurred before the plastic operation and therefore could not be directly attributed to the latter procedure. It is noteworthy that the 3 patients presented evidence of intracranial complications at the time of their first admission to the hospital.

The first patient, aged 25, presented meningeal signs, stiffness of the neck and Kernig's sign. The lumbar puncture showed fluid under a pressure of 270, with 890 cells, 27 per cent of which were polymorphonuclears. She presented bilateral papilledema also. On resection of the frontal bone an extradural abscess was found. Four days after the operation she had her first generalized convulsion.

The second patient, aged 13, was admitted after jacksonian epilepsy on the left side. When the frontal bone was resected granulations covering the dura and an extradural abscess were found. After the operation the lumbar puncture showed fluid under a pressure of 180, with 13 cells, all lymphocytes. Examination of the fundi revealed bilateral papilledema.

The third patient, aged 22, showed stiffness of the neck without Kernig's sign. On lumbar puncture turbid fluid under a pressure of 200 was found, with 810 cells, 70 per cent of which were polymorphonuclears. The dura, after resection of the frontal bone, showed an area covered with granulations, although no definite extradural abscess was found. One month later, an abscess of the frontal lobe was localized and drained. Eighteen months later, the patient had her first general convulsive seizure. The attack of epilepsy occurred, however, before the plastic closure, the latter procedure having been purposely delayed after the appearance of these intracranial complications.

It is possible, therefore, to draw three general conclusions: first, that the convulsions were not due to the operative reconstruction; second, that the convulsions occurred in cases in which definite pathologic changes in the intracranial area were presented, and, third, that the soft tissue should not be closed until all suppuration has ceased and until the possibility of intracranial complications has been eliminated.

CONCLUSIONS

The adoption of a radical procedure in dealing with osteomyelitis of the frontal bone has brought up the problem of secondary plastic repair of the deformity.

Plastic reconstruction should be considered only when suppuration and intracranial complications have been eliminated.

Except in cases of children, bony regeneration of the operative defect has not been observed.

The reconstruction is done in two stages. The first stage is a closure of the defect in the soft tissue. The second stage is the repair of the bony defect with bone grafts.

Epilepsy could not be attributed to the plastic operation and was observed only in cases in which intracranial complications were presented earlier, at or near the time of the operation for osteomyelitis.

ABSTRACT OF DISCUSSION

DR. HARRIS P. MOSHER, Boston: This is a timely paper. It rounds out the report of the work on fulminating osteomyelitis of the frontal bone complicating infection of the frontal sinus which my associates and I have been doing for the past eight years at the Massachusetts Eye and Ear Infirmary. Why we see only the fulminating type, following swimming, I do not know. It sounds like an alibi to say that the severity of the infection of osteomyelitis may differ in different parts of the country. This, however, happens in infection of the mastoid. The epidemiology of osteomyelitis has not been worked out; neither has sulfanilamide or sulfapyridine entered the picture, as it will do in our next cases.

In the operative management of our cases we have settled down to a procedure which is practically routine, the only one which has given us increasingly better results and kept us from floundering about as we used to do. I am still waiting for some one to tell me how to determine at the beginning of an infection whether it will localize and the bone sequestrate or whether it will spread like wild fire. I am not advising a radical, deforming operation just from the lust for surgical procedure; until I get the information just mentioned I shall not trim my sails.

These frank statements will, I hope, dam up a little of the flood of oratory on the type of operation to be used, which I know from past experience is easily started. The type of operation is certainly not the main topic of the paper under discussion.

The operation for osteomyelitis of the frontal bone is simple. It is really an encircling operation. The only hard thing about it is to make up one's mind to do it and do it early. I have given up appeasement.

The radical operation for osteomyelitis is really a three stage operation. The first stage consists of the removal of the diseased bone, and the other two comprise the plastic repair of the deformity left by the first. No one likes to leave a patient markedly deformed after operation, as would be the case without the plastic repair which is the subject of Dr. Kazanjian's orderly, well reasoned, informative paper. On the basis of an impressive number of cases, he has given his present, up-to-date technic, which he has naturally bettered as the number of his cases has increased.

I should like to emphasize three points dealt with in the paper—the time of closure, the regeneration of bone and the occurrence of convulsions before or after the plastic closure.

The surgeon should err on the safe side in determining the time of closure. We have had trouble a few times with small sequestrums or the opening up of the lower part of the wound in the middle line when the closure was made before three months.

As to regeneration of bone, we have not been able to duplicate the experience of Furstenberg. In most of his reported cases the operative defect filled in with new bone. This is, of course, much to be desired. Dr. Kazanjian has had but 2 cases in which this has occurred, and both the patients were children. From the literature one gets the impression that the flat bones of the skull do not regenerate.

It is the common belief that bone regenerates chiefly from the periosteum. In talking with Dr. Kazanjian, I have gathered that he feels that we maltreat the periosteum by the hurried removal of bone and by the disturbance of frequent dressing and frequent inspection of the edges of the defect for progressive necrosis of the bone. I feel that there is something in this view and am in favor, since there is a wide open wound, of determining such a happening by roentgen examination, even though roentgen evidence is shown late. In other words, one can afford to wait a little if by so doing one gives the periosteum a better chance to bring about regeneration of bone.

It is comforting to learn of the 104 cases of successful transplantation of bone grafts to correct deformity reported by Delangenière. The size of the bone grafts used by Dr. Kazanjian is a bit startling. He gives convincing reasons why the bone graft should not be used at the original operation, however great and natural is the temptation.

It is likewise tempting to try filling the operative defect with the bone which has been removed at operation after sterilizing it or to use animal bone. However, it hardly seems worth while when there is plenty of live bone in the patient's nearby tibia from which an autogenous graft can be taken.

Convulsions may occur before or after the plastic closure. Dr. Kazanjian naturally would like to have them all occur before the plastic repair and cites 3 cases in which they did so occur. Convulsions in connection with osteomyelitis are due to scar tissue or to cysts of scar tissue.

Osteomyelitis is an ideal disease to cause scar tissue. The epileptic attack, so-called, are pressure convulsions like those of typical jacksonian epilepsy. Much as one dislikes to have them occur, they should cause no surprise. The wonder is that they do not occur more often. If they persist after a reasonable period of rest and observation the treatment is the obvious one of opening the incision and dissecting out the cyst or removing the offending scar if it can be located.

No surgeon is justified in assuming responsibility for a patient with fulminating osteomyelitis of the frontal bone unless he fully realizes what the frequent complications are and knows that he is dealing with one of the most fatal of surgical diseases. In no other part of the body has partial operation or watchful waiting more often proved disastrous.

The general surgeon is behind the times in treating this condition, as he is in treating deep abscess of the neck.

Dr. Vilray P. Blair, St. Louis: I think the last two remarks were bait thrown out to me, but I am not taking it. In the time allotted, I will stick to what I was asked to discuss—the treatment of a defect in the skull.

I was much interested in Dr. Kazanjian's use of the osteoperiosteal graft from the tibia. I have never performed that operation. My associates and I have observed defects of various sizes and from various causes, and in dealing with all there are two things at which to aim: One is to close the defect for the better function of the contents of the skull, and the other is to improve the appearance.

Some small defects we have filled with cartilage, and for larger ones we prefer the plan of chiseling off an adjacent thin layer of the outer table. We have roofed over large defects with rafters of closely placed ribs, the ends of which

were notched to fit shiplap at the edge of the defect, their natural curve partially restoring the dome shape. A large loss of the vertex, such as formerly was not infrequently encountered after "decompression operations" or epilepsy, is both crippling and unsightly. In such cases the Naffziger plan of transplanting a fitted corresponding piece of boiled calvarium is ideal if the graft is biologically acceptable to the tissues, and there is no real reason why the particular piece of bone could not be tried out in the patient's tissue before the real operation is undertaken.

For improving the appearance also I think one can use cartilage, and we have come to use fascia lata rather than the Eitner dermal grafts, because in our hands the former have proved more certain to take.

In a number of cases, cartilage smoothed out with fascia lata helps tremendously in giving the restoration desired.

Granting, for the sake of the present discussion, that Dr. Mosher is right in his premise that radical operation is the way to treat osteomyelitis, I think he is fortunate to have Dr. Kazanjian to cover up for him, which he has done beautifully.

THE GALVANIC REACTION IN GUINEA PIGS

III. THE REACTION FOLLOWING SECTION OF THE EIGHTH NERVE

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In two previous communications [1] we have discussed the galvanic reaction in normal and in unilaterally and bilaterally labyrinthectomized guinea pigs. The present study has to do with the galvanic reaction in guinea pigs in which the eighth nerve on one side was destroyed.

Dohlman [2] experimented with the galvanic reaction of animals in which the vestibular nerves were sectioned intracranially or in which the vestibular ganglion had been removed. He concluded that elicitation of galvanic nystagmus is dependent on integrity of the cells in the vestibular ganglion and their central connections. This conclusion is in agreement with that of Blau,[3] who found in cats, guinea pigs and an ape that nystagmus could still be elicited with the galvanic current when the labyrinth was completely destroyed. He proved by microscopic examination of the temporal bone that Scarpa's ganglion cells and the eighth nerve were still intact in these animals. Malassez,[4] after conducting examinations of deaf-mutes, concluded that the integrity of the semicircular canals is essential for postrotatory nystagmus but that the galvanic reaction can be obtained if a part at least of the vestibular nerve is still intact. Northington and Barrera,[5] working with monkeys,

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1. Buchanan, A. R., and Ladd, L. D.: The Galvanic Reaction in Guinea Pigs: I. The Normal Galvanic Reaction, Arch. Otolaryng. **29**:124-135 (Jan.) 1939; II. The Reaction Following Labyrinthectomy, ibid. **29**:136-145 (Jan.) 1939.

2. Dohlman, G.: Experimentelle Untersuchungen über die galvanische Vestibularisreaktion, Acta oto-laryng. (supp.) **8**:1-48, 1929.

3. Blau: Experimentelle Studien über den galvanischen Nystagmus, Ztschr. f. Ohrenh. (supp.) **69**:6-7, 1913.

4. Malassez, J.: Excitation mécanique et éléctrique du vestibule chez les sourds-muets, Compt. rend. Soc. de biol. **89**:1256-1257, 1923.

5. Northington, P., and Barrera, S. E.: Induced Nystagmus in Monkeys Following Peripheral Vestibular Lesions, with Clinical Correlations, Laryngoscope **47**:729-754 (Oct.) 1937.

came to the conclusion that anatomically or functionally intact labyrinths are not necessary for the production of nystagmus by the galvanic current, but they found that an intact eighth nerve appears to be necessary for eliciting the response. In their opinion, a positive galvanic response (nystagmus) in the presence of a negative caloric response on the side on which there is vestibular dysfunction indicates that the eighth nerve on that side is not degenerated.

PROCEDURE

In the present study we destroyed the eighth nerve on the left side in 13 guinea pigs. The method employed for destruction of the nerve consisted of making a small drill hole through the skull, directly above the internal auditory meatus. An insulated electrode was inserted through this opening and forced down between the cerebellum and the lateral wall of the cranium. A right-angled projection at the distal end of the electrode was inserted into the internal auditory meatus, the tip of which was devoid of insulation. Orientation was simple, and a stop on the electrode indicated the approximate distance downward from the opening in the skull to the internal auditory meatus. Once oriented in the internal auditory meatus, the electrode could be held rigidly in place while a galvanic current of about 10 milliamperes was allowed to flow through the electrode. An indifferent electrode was attached to some point on the surface of the animal. The success of the procedure was evident at once, since the eyes immediately deviated toward the side on which the lesion was present, the right eye upward and the left downward. Since we used ether anesthesia for the procedure, the animals revived quickly, and spontaneous nystagmus toward the right became evident immediately. The animals also showed rotation of the head and concavity of the vertebral column toward the left, and they rolled violently in the same direction. Absence of the corneal reflex in the left eye indicated destruction of the seventh nerve, which was anticipated because of the close relation of this nerve to the eighth nerve in the internal auditory meatus.

Before operation a kymographic record of the galvanic falling reaction in each of these animals was obtained. It was ascertained also that the eyes moved normally in response to changes in the position of the animal in space (head up, head down and right and left lateral positions). Records of the galvanic falling reaction were made on the fourth and the fifteenth postoperative day. Caloric tests were carried out on the fifth and the sixteenth postoperative day. The methods utilized in testing the galvanic reaction have been discussed in previous papers.[1] The methods for testing the caloric reaction have been discussed in a previous communication by one of us (A. R. B.).[6]

On the fifteenth or the sixteenth postoperative day the animals were killed, after perfusion with neutral 10 per cent solution of formaldehyde, and the brains were fixed according to the method of Swank.[7] They were embedded in pyroxylin, sectioned and studied microscopically in order to determine whether the eighth nerve was completely degenerated.

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6. Buchanan, A. R.: Eye Deviation and Nystagmus in Guinea Pigs with Lesions of the Cerebellum and Brain Stem, Laryngoscope **47**:874-900 (Dec.) 1937.

7. Swank, R.: Marchi Type Stains Counterstained with Cresyl Violet, Anat. Rec. (supp.) **61**:63 (March) 1935.

RESULTS

Protocol.—A protocol of a typical experiment follows. A guinea pig was tested for reactions to the galvanic current on Oct. 13, 1938, at which time 3 milliamperes of current was sufficient to elicit a reaction from either ear. Operation was performed on this animal on October 18. After the operation, which was performed with ether anesthesia, the animal rolled toward the left (the side on which the operation had been done); its eyes deviated toward the left, and there was spontaneous nystagmus to the right. The head was turned and rotated toward the left. There was also absence of the corneal reflex in the left eye, indicating paralysis of the seventh nerve.

October 19: All the symptoms of destruction of the eighth and of the seventh nerve seen immediately after the operation were still present.

October 21: All the symptoms were still present except the rolling toward the left. Galvanic tests carried out on this date (fourth postoperative day) showed an increase in the number of paradoxic reactions on both sides over that seen preoperatively, the greater number being elicited from the ear on the experimental side. On the normal side there were two paradoxic reactions per 10 stimuli, and on the side of operation, 5 such reactions. The currents required were also increased on both sides, being 5 milliamperes on the normal side and 15 milliamperes on the side of operation.

October 22: The deviation of the eyes persisted, but there was no longer spontaneous nystagmus. A caloric examination carried out on this date (fifth postoperative day) gave results as follows: Stimulation of the right ear gave nystagmus upward and forward in the right eye and downward and backward in the left. This nystagmus was easily elicited and had a normal duration. Caloric stimulation of the left ear gave a slight nystagmus in the opposite direction. The fact that there was some nystagmus from caloric stimulation of the left ear probably indicates that degeneration of the nerve was incomplete.

November 1: Rotation of the head to the left, deviation of the eyes to the left and paralysis of the left seventh nerve were still present. A galvanic test carried out on this date (fifteenth postoperative day) showed no paradoxic reaction from the normal ear, while there were still 5 such reactions per 10 stimuli from the opposite ear. The currents required were 3.5 milliamperes on the normal side and 19 milliamperes on the side of operation.

November 2 (sixteenth postoperative day): Caloric examination gave results as follows: Irrigation of the right ear elicited nystagmus forward and upward in the right eye and downward and backward in the left eye. No nystagmus could be elicited by stimulation of the left ear. The animal was killed. Neutral solution of formaldehyde was injected into the brain, and the organ was fixed according to the method of Swank.[7] Histologic study of the brain showed degeneration of the vestibular division of the eighth nerve on the left side. The degenerated fibers could be traced into the vestibular nuclei. Interestingly, some of these fibers apparently ran directly into the cerebellum. This supports the opinion of Lorente de Nó[8] and others. There was also considerable degeneration in the trapezoid body, due apparently to direct injury to the ventral cochlear nucleus.

Paradoxic Reactions.—Preoperatively, as has been true of each series of normal guinea pigs examined by this method, some paradoxic reactions were observed; of the total number of reactions observed in 13 animals, 6.3 per cent of those

8. Lorente de Nó, R.: The Anatomy of the Eighth Nerve: Central Projection of the Nerve Endings of the Internal Ear, Laryngoscope **43**:1-38 (Jan.) 1933.

elicited by stimulation of the right ear and 6.1 per cent of those elicited by stimulation of the left ear were in the wrong direction. On the fourth postoperative day there was an increase in the number of paradoxic reactions from the ears on both the surgically treated and the normal side. The greater number of paradoxic reactions, however, were observed on the side which had been operated on, averaging 5 paradoxic reactions per 10 stimuli, or 50 per cent; on the normal side there was an average of 3.63 paradoxic reactions per 10 stimuli, or a percentage of 35.3.

On the fifteenth postoperative day the number of paradoxic reactions was considerably less than on the fourth day, averaging 2.25 reactions per 10 stimuli on the normal side and 1.12 reactions on the side of operation. The percentage of paradoxic reactions on the normal side was 22.5, and on the side of operation, 11.2. It will be noted that on the fourth postoperative day, while paradoxic responses were increased on both sides, more were obtained from the side of operation. On the fifteenth day, on the other hand, more paradoxic responses were obtained from the normal than from the experimental side. Only 1 animal of the 13 showed

Summary of the Results of Galvanic Stimulation of Thirteen Animals Before and After Section of the Eighth Nerve *

Average Number of Paradoxic Reactions per Ten Stimuli (13 Animals)						Average Amount of Current, Milliamperes (13 Animals)					
		Postoperative						Postoperative			
Preoperative		4 Days		15 Days		Preoperative		4 Days		15 Days	
Right	Left	Unop.	Op.	Unop.	Op.	Right	Left	Unop.	Op.	Unop.	Op.
0.63	0.61	3.53	5.00	2.25	1.12	2.65	2.61	5.92	9.15	2.81	6.76
6.3%	6.1%	35.3%	50.0%	22.5%	11.2%						

* In this table, "right" indicates the electrode placed in the right ear; "left," the electrode placed in the left ear; "Unop.," the electrode placed in the ear on the side opposite the nerve section, and "Op.," the electrode placed in the ear on the side on which the nerve was sectioned.

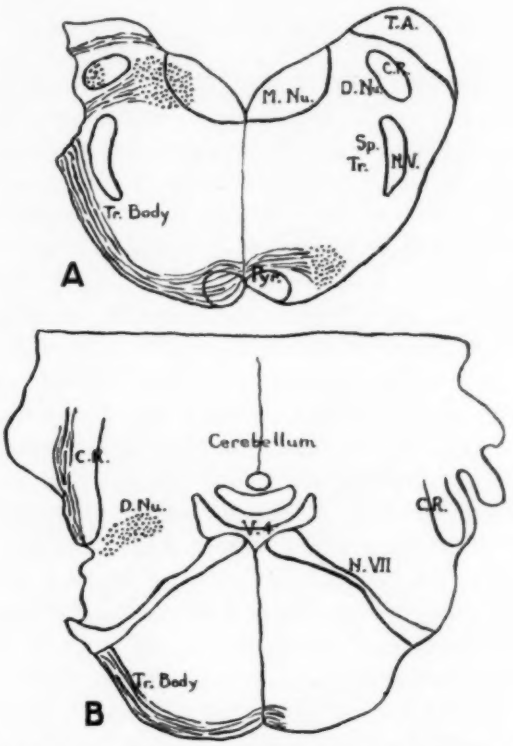
more paradoxic reactions on the experimental than on the normal side on the fifteenth day.

Amount of Current Necessary for Responses.—The average amount of current required for a galvanic reaction in this group of animals before operation was 2.65 milliamperes for the right ear and 2.61 milliamperes for the left ear. Postoperatively the amount of current required for a reaction was considerably increased. On the fourth day an average of 5.92 milliamperes was required for reactions from the ear on the normal side and an average of 9.15 milliamperes was required on the experimental side. By the fifteenth day the current required on the normal side was reduced to within normal limits, with an average of 2.81 milliamperes. On the side of operation it still required an average of 6.76 milliamperes to produce a reaction.

Sign of More Effective Stimulus.—Before operation anodal and cathodal stimulation of the ears were equally effective in producing a response in 34 per cent of cases; anodal stimulation was the more effective in 34 per cent, and cathodal stimulation in the remaining 32 per cent. On the fourth postoperative day anodal and cathodal stimulation were equally effective in 27 per cent of the cases; anodal stimulation was the more effective in 27 per cent, and cathodal stimulation in the

remaining 46 per cent. On the fifteenth postoperative day anodal and cathodal stimulation were equally effective in 13 per cent of the cases; anodal stimulation was the more effective in 31 per cent, and cathodal stimulation in the remaining 56 per cent.

As with the labyrinthectomized animals, cathodal stimulation of the ear tended to be more effective than anodal stimulation after operation. Before operation there was an almost equal distribution of the cases in which anodal and cathodal stimulation were equally effective, those in which anodal stimulation was the more effective and those in which cathodal stimulation was the more effective. After



Drawings of the brain stem. *T. A.* indicates the tuberculum acusticum; *C. R.*, the corpus restiforme; *D. Nu.*, Deiters' nucleus; *M. Nu.*, the medial vestibular nucleus; *Sp. Tr. N. V*, the spinal tract of the fifth nerve; *Tr. Body*, the trapezoid body (degenerating); *V. 4*, the fourth ventricle; *N. VII*, the seventh nerve; *Pyr.*, the pyramid.

the operation the cases in which cathodal stimulation was the more effective about equaled in number those in which the other two conditions held.

Sections of the brain stem of 1 of the guinea pigs are illustrated in the accompanying figure. It represents the picture in each of the 13 animals included in this report. In *A* the degenerated fibers of the vestibular division of the eighth nerve can be seen terminating in the vestibular nuclei. The trapezoid fibers from the ventral cochlear nucleus are also shown running ventrally and medially to cross the midline and form the lateral lemniscus. The degeneration of the trapezoid

body is accounted for by direct injury to the ventral cochlear nucleus, which is also clearly evident in the figure. In *B* the seventh nerve can be seen emerging from the brain stem, which, of course, indicates the level at which the vestibular nerve enters; it can be seen that a portion of the brain stem was actually destroyed at the point of entrance of the nerve. Even though the degeneration of the fibers of the eighth nerve terminating in the vestibular nuclei on the left side were not evident, the actual destruction of tissue peripherally at the point of entrance of the eighth nerve would prove beyond doubt that the vestibular division of the eighth nerve was completely severed.

COMMENT AND SUMMARY

The galvanic falling reaction has been tested in guinea pigs with unilateral destruction of the eighth (auditory and vestibular) and the seventh nerve. Contrary to our expectations and contrary to the results obtained by some previous investigators, the reaction was still elicitable in these animals. Previous investigators, however, including Dohlman,[2] Blau,[3] Malassez[4] and Northington and Barrera,[5] studied not the galvanic falling reaction but galvanic nystagmus. We have not made careful observations on nystagmus as elicited by the galvanic current, but further studies which will include a study of both phenomena are necessary.

In our study of the galvanic reaction of unilaterally labyrinthectomized guinea pigs it was found that paradoxic reactions occurred more frequently on the side which was not operated on than on the side of the labyrinthectomy. In the animals in which the eighth nerve was destroyed unilaterally it was found that paradoxes occurred on stimulation of the ears on both sides; on the fourth postoperative day the average percentage of paradoxic reactions was 35.3 on the normal side and 50 on the side of operation; on the fifteenth day the paradoxic reactions occurred about twice as frequently on the normal as on the experimental side, the percentage being, respectively, 22.5 and 11.2.

In the unilaterally labyrinthectomized animals greater amounts of current were required on both sides, but considerably more was required on the labyrinthectomized side. This was true on the fourth postoperative day in the animals with the eighth nerve destroyed, when the average current required on the normal side was 5.92 milliamperes, as compared with 2.65 milliamperes on the same side before operation, and 9.15 milliamperes on the experimental side, as compared with 2.61 milliamperes on the same side before operation. On the fifteenth postoperative day, however, the average current required on the normal side was practically the same as before operation (2.81 milliamperes). On the experimental side 6.76 milliamperes was required to elicit a reaction.

As in the labyrinthectomized animals, cathodal stimulation of the ears seemed to be more effective in the surgically treated than in the normal guinea pigs.

Differentiation between disease of the labyrinth and disease of the eighth nerve would be impossible on the basis of this test, since the reactions in the wrong direction increased on both sides in both types of animals and in about the same proportion. The variations in the strength of current required were essentially the same in the two conditions.

The fact that the galvanic falling reaction persisted after destruction of the eighth nerve indicates that some other mechanism must function in this reaction. What this mechanism may be is difficult to say. It may be that the tenth nerve, particularly through the division known as Arnold's nerve, which furnishes the sensory supply to the external auditory meatus, may transmit the stimulus to the proper brain centers, whatever they may be. Other nerves which must be considered include, of course, the fifth, the ninth, the tenth and the eleventh cranial nerve, and possibly the upper cervical nerves. It will be necessary to devise some means of eliminating these nerves one at a time and, it seems to us, in one and the same animal in order to obtain definite information as to the part which they may play in the galvanic falling reaction.

THE GALVANIC REACTION IN GUINEA PIGS

IV. THE REACTION IN ANIMALS WITH UNILATERAL LESIONS IN THE BRAIN STEM

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Previous reports have described the galvanic reaction in guinea pigs with unilateral labyrinthectomy and with unilateral destruction of the eighth nerve.[1] It has been shown, contrary to the findings in human subjects reported by Blonder,[2] that the reaction is still elicitable after destruction of the labyrinth, although requiring a considerably stronger current. This is in agreement with the findings reported by Blau [3] and others, including those in the recent report of Northington and Barrera.[4] After destruction of the eighth nerve the galvanic falling reaction could still be elicited, but, again, a stronger current was required than for normal animals. Dohlman,[5] Northington and Barrera [4] and others have found that galvanic nystagmus cannot be elicited after destruction of the eighth nerve.

The nervous structures involved in the galvanic falling reaction seem, logically, to include: (1) the labyrinth; (2) the vestibular division of the eighth nerve; (3) the vestibular nuclei, and (4) the descending pathways from the vestibular nuclei, including the vestibulospinal tract and the medial longitudinal fasciculi. Any explanation why a pathway so constituted should be responsible for an almost constant falling reaction toward the anode and away from the cathode when galvanic current is allowed to flow through the ear is, so far as I know, still lacking. Since

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1. Buchanan, A. R., and Ladd, L. D.: The Galvanic Reaction in Guinea Pigs: II. The Reaction Following Labyrinthectomy, Arch. Otolaryng. **29**:136-145 (Jan.) 1939; III. The Reaction Following Section of the Eighth Nerve, ibid., this issue, p. 113.

2. Blonder, E. J.: Galvanic Falling in Clinical Use, Arch. Neurol. & Psychiat. **37**:137-141 (Jan.) 1937.

3. Blau: Experimentelle Studien über den galvanischen Nystagmus, Ztschr. f. Ohrenh. (supp.) **69**:6-7, 1913.

4. Northington, P., and Barrera, S. E.: Galvanic Nystagmus Reaction in the Monkey, Am. J. Physiol. **120**:703-704 (Oct.) 1937.

5. Dohlman, G.: Experimentelle Untersuchungen über die galvanische Vestibularisreaktion, Acta oto-laryng. (supp.) **8**:1-48, 1929.

the galvanic falling reaction can be elicited after destruction of the labyrinth or after destruction of the eight nerve, the hypothesis seems to be in error in describing either the first or the second or both the first and the second portion of the pathway; if it is not in error, other structures must be accessory to these portions. The latter seems to be true, since elimination of the labyrinth or of the eighth nerve does not eliminate the reaction but does necessitate the use of larger amounts of current. Furthermore, the tendency of animals with either of these structures destroyed to react toward the anode is considerably less constant.

It may be that a study of the reaction following destruction of the third or central portion of the hypothetic pathway (the vestibular nuclei) is not indicated until the peripheral pathway has been more definitely determined. Nystagmus, as elicited by galvanic, caloric and rotatory stimulation has, however, been studied by Dohlman [5] after extirpation of the triangular (medial) vestibular nucleus. He found that post-rotatory and caloric nystagmus could still be elicited but that galvanic nystagmus was no longer elicitable. Blonder and Davis [6] studied the galvanic reaction in patients with intracranial tumors and found that the average amount of current required to produce falling in those with supratentorial lesions was less than that required in those with infratentorial lesions. Since there has been an attempt to utilize the reaction clinically, in the diagnosis of lesions in the brain stem, it seems that the results obtained in a series of guinea pigs with lesions in the brain stem are significant.

PROCEDURE

Kymographic records of the galvanic reactions in 15 normal guinea pigs were made, and the animals were subsequently operated on. Lesions were placed in the brain stem by means of a Horsley-Clarke stereotaxic instrument; all the lesions were unilateral and all were on the left side. In 6 animals the lesions involved the lateral vestibular nucleus, or nucleus of Deiters (series 1, table); in 7 animals there was destruction in the medial vestibular nucleus (series 2, table); in 4 animals there was involvement of the descending vestibular nucleus (series 3, table). The use of the horizontal electrode obviated any injury to the cerebellum.

Postoperative tests of the galvanic reaction were carried out on the fourth and on the fifteenth day. In all the galvanic tests local nupercaine anesthesia was used at the points of application of the electrodes. The detailed technic of the galvanic determinations has been reported previously.[7]

Nine of the animals included in the table were operated on a second time at the end of the fifteenth days' observation, a second lesion being placed on the same

6. Blonder, E. J., and Davis, L.: The Galvanic Falling Reaction in Patients with Verified Intracranial Neoplasms, J. A. M. A. **107**:411-412 (Aug. 8) 1936.

7. Buchanan, A. R., and Ladd, L. D.: The Galvanic Reaction in Guinea Pigs: I. The Normal Galvanic Reaction, Arch. Otolaryng. **29**:124-135 (Jan.) 1939.

Animal No.	Site of Lesion*	Number of Paradoxic Reactions per 10 Stimuli						Amount of Current in Milliamperes					
		Preoperative		Postoperative				Preoperative		Postoperative			
				4th Day		15th Day				4th Day		15th Day	
		Electrode on Right Side	Electrode on Left Side	Electrode on Intact Side	Electrode on Injured Side	Electrode on Intact Side	Electrode on Injured Side	Electrode on Right Side	Electrode on Left Side	Electrode on Intact Side	Electrode on Injured Side	Electrode on Intact Side	Electrode on Injured Side
						Series I							
114	Deiters' nucleus; inferior cerebellar peduncle; eighth nerve	0	0	4.4	5	+	+	4	2.5	2.5	5	+	+
115	Deiters' nucleus (slight); inferior cerebellar peduncle	0	0	4.4	4.7	0.77	7	2.5	4	3	10	3	3
133	Deiters' nucleus; spinal tract and nucleus of the fifth nerve	0	0	0	2	3	0	2.5	3	2	2	2	2
135	Deiters' nucleus (slight); descending nucleus; lateral cuneate nucleus; spinal nucleus of the fifth nerve	0	1.4	5	0.77	+	+	3	3	4	4.5	+	+
129	Deiters' nucleus; medial nucleus	0	0	0	0	0	0	2.5	2.5	4	5.5	3.5	3
130	Deiters' nucleus; medial nucleus; uncinate fasciculus (of Russell); genu of the seventh nerve	2	0	0	0	+	+	2	2	2.5	2.5	+	+
	Average	0.33	0.23	2.3	2.08	1.26	2.33	2.75	2.83	3.00	4.91	2.83	2.66
	Percentage	3.3	2.3	23	20.8	12.6	23.3						
						Series II							
118	Medial nucleus (slight); medial reticular formation	0	0	2.5	0.9	0	1	2.5	2.5	3	3	2.5	2.5
119	Medial nucleus (slight); medial reticular formation	0	0	2	3.7	0	0.71	5	5	4	4	2	2.5
120	Medial nucleus; medial reticular formation	0	0	+	+	+	+	?	?	+	+	+	+
121	Medial nucleus	0	0	5.7	5	+	+	2.5	2.5	5	5	+	+
134	Medial nucleus (slight); reticular formation	0	0	0	0	4.1	0	3	4	3	2.5	2	3
129	Medial nucleus; Deiters' nucleus	0	0	0	0	0	0	2.5	2.5	5	5	+	+
132	Medial nucleus; spinal nucleus of the fifth nerve	0	0	0.83	0	2.8	0	3.5	3	4	2	5	2.5
	Average	0	0	1.84	1.60	1.38	0.34	3.16	3.25	3.83	3.66	3.00	2.70
	Percentage	0	0	18.4	16.0	13.8	3.4						
						Series III							
117	Descending nucleus	0	0	0	0.77	0	5	3	4	2.5	5	4	4
128	Descending nucleus	0	0	5	5	0	0	3	3	10	7	2	2
135	Descending nucleus; Deiters' nucleus (slight); lateral cuneate nucleus; spinal nucleus of the fifth nerve	0	1.4	5	0.77	+	+	3	3	4	4.5	+	+
136	Descending nucleus (slight); lateral reticular formation; eighth nerve	0	0	3	1.6	+	+	2.5	3.5	2.5	2.5	+	+
	Average	0	0.36	3.25	2.03	0	2.5	2.87	3.37	4.75	4.75	3.00	3.00
	Percentage	0	3.6	32.5	20.3	0	25						
	General average	0.12	0.16	2.36	1.89	1.07	1.37	2.94	3.12	3.75	4.42	2.75	2.95
	Percentage	1.2	1.6	23.6	18.9	10.7	13.7						

side of the brain stem. Postoperative observations were made on these animals in exactly the same manner as after the first operation.

On termination of the postoperative study, the animals were killed, and the brains were removed, fixed and studied by means of the Weil [8] and cresyl violet [9] stains, which facilitated accurate localization of the lesions.

RESULTS

Paradoxic Reactions.—The number of paradoxic reactions per ten stimuli for the entire group of animals before operation averaged 0.12 on stimulation of the right ear and 0.16 on stimulation of the left ear (table); the respective percentages were 1.2 and 1.6. The percentage of paradoxic responses on the fourth day after operation was 23.6 on the intact side and 18.9 when the electrode was in contact with the ear on the side of the lesion. On the fifteenth postoperative day the respective percentages were 10.7 and 13.7.

If one considers only the 6 animals with lesions involving Deiters' nucleus (series 1, table), the preoperative paradoxes occurred in 3.3 per cent of cases when the right ear was the site of stimulation and in 2.3 per cent when the left ear was stimulated. On the fourth postoperative day 23 per cent of the reactions from the ear on the intact side and 20.8 per cent of the reactions from the ear on the side of the lesion were in the wrong direction. Fifteen days after operation the respective percentages were 12.6 and 23.3.

Separate consideration of the 7 animals with involvement of the medial vestibular nucleus reveals that there were no paradoxic reactions in this group before operation (series 2, table). On the fourth postoperative day the intact and the injured side showed respectively 18.4 per cent and 16 per cent paradoxic reactions. On the fifteenth day the respective percentages were 13.8 and 3.4.

Only 1 of the 4 animals having involvement of the descending nucleus gave any paradoxic responses before operation, this animal (135) reacting in the wrong direction 14 per cent of the time. The average percentage of paradoxic reactions in this group (series 3, table) was 0 on the right and 3.6 on the left. Four days after operation the percentages were 32.5 on the intact side and 20.3 on the side of the lesion. On the fifteenth day there were no paradoxic reactions from the intact side, and 25 per cent of the reactions from the side of the lesion were in the wrong direction.

Amount of Current in Milliamperes.—The average number of milliamperes required for elicitation of the falling reaction before operation

8. Weil, A.: A Rapid Method for Staining Myelin Sheaths, Arch. Neurol. & Psychiat. **20**:392-393 (Aug.) 1928.

9. Tress, G., and Tress, M.: A Modification of the Cresyl Violet Technic for Staining Nerve Cells, Stain Technol. **10**:105-106, 1935.

in this group of animals was 2.94 for the right ear and 3.12 for the left ear. There was a slight increase in the amount of current required on the fourth day after operation (3.75 milliamperes on the intact side and 4.42 on the side of the lesion), but the increase was equal or practically equal on the injured and the intact side, this being true of the general average and also of the average for each of the three series of animals (table). On the fifteenth day after operation the average requirement of current was within normal limits on both the injured and the intact side (2.75 and 2.95 milliamperes).

Results of Second Operation.—As previously stated, 9 of the animals included in this report were operated on a second time, following the first fifteen days' observations, and a second lesion was placed on the same side of the brain stem; a detailed discussion of the areas involved by these supplementary lesions will be omitted. There was, however, approximately twice as much destruction in the left half of the brain stem of these animals as existed after the first operation. In these, on the fourth postoperative day, 36.3 per cent of the reactions from the ear on the intact side were in the wrong direction, or paradoxic, and 19.8 per cent of those from the side of the lesion were in the wrong direction. The requirement of current was 5.22 milliamperes on the intact side and 4.44 on the side of the lesion. On the fifteenth day there were still about twice as many paradoxic reactions from the ear on the intact side as from that on the side of the lesion, the respective percentages being 13.5 and 6.8. At this time the requirement of current was again well within the normal range, being 2.25 milliamperes on the intact side and 3 on the side of the lesion.

Sign of the More Effective Stimulus.—Careful analysis has shown that there was no tendency for the galvanic current to be more effective when either the cathode or the anode was in contact with the ear. In this respect, the animals differed from those with lesions in the labyrinth or the eighth nerve, in which cathodal stimulation appeared to be more effective than anodal.

DISCUSSION

Obviously, the galvanic falling reaction might serve a most useful purpose clinically, if lesions in the brain stem could be localized to one or the other side by means of it. If the vestibular nuclei form a link in the pathway which effects the galvanic falling reaction, it seems that one might expect a change in the reaction to follow unilateral destruction of the nuclei or their connections. Unfortunately, however, the differences in the requirement of current on the injured and the intact side were insignificant. Furthermore, the number of paradoxic reactions per ten stimuli was not significant in lateral localization of the lesion, being slightly greater on the intact side on the fourth postoperative day

and slightly greater on the side of the lesion on the fifteenth day, except in the animals which were studied after a second lesion had been placed in the brain stem. In the latter series of guinea pigs, the number of paradoxic reactions from the ear on the intact side was approximately twice as great as the number from the side of the lesion on both the fourth and the fifteenth postoperative day. The results in these animals seem to indicate a tendency toward an increase in paradoxic reactions from stimulation of the ear on the intact side, a tendency similar to that in guinea pigs with lesions in the labyrinth or the eighth nerve. The electrolytic lesions produced in these animals are, of course, static if not actually regressive; it seems probable that an increase in paradoxic reactions might regularly accompany expanding lesions. If more massive lesions were compatible with postoperative recovery in guinea pigs, an increase in the number of paradoxic reactions, even on the fifteenth postoperative day, might probably be observed after a single operation. The placing of a second lesion at a later date should bring about essentially the same effect.

The fact that paradoxic reactions occur more frequently on both sides, but especially on the intact side, in guinea pigs with lesions in the labyrinth, the eighth nerve or the vestibular nuclei, destroys the value of the test in differentiating any one of these conditions from the other two. The amount of current required does become significant in differentiation; in the animals with central lesions, increase in the requirement of current was minimal on the fourth day and absent on the fifteenth; in those with destruction of the labyrinth there was a considerable increase in the amount of current required on the side of the lesion; section of the eighth nerve still further increased the amount of current required to elicit the galvanic falling reaction.

Dohlman's conclusions [5] were not verified, although it must be noted that they were based on studies of galvanic nystagmus rather than on the galvanic falling reaction. Caloric and postrotatory nystagmus could be elicited in each of the 7 animals in series 2, table, which had lesions involving the medial vestibular nucleus. The galvanic falling reaction also was elicitable in these animals, although somewhat modified as shown in the table and discussed.

Caloric and rotatory tests produced nystagmus in all the guinea pigs included in the table. Modifications in the caloric and postrotatory nystagmus over that seen in normal animals will be discussed in another communication.

SUMMARY

1. Paradoxic reactions were increased on stimulation of the ears on both the injured and the intact side in guinea pigs with electrolytic lesions in the left half of the brain stem. When the lesions were exten-

sive, the number of paradoxic reactions from the intact side was about twice as great as that from the side of the lesion, a condition which was found to exist also in animals with lesions in the labyrinth or the eighth nerve.

2. Significant increases in the number of milliamperes of current required to elicit galvanic falling reactions on stimulation of either ear were not observed, even in animals having extensive destruction of the vestibular nuclei or the reticular formation.

3. The galvanic current did not tend to be more effective when either the cathode or the anode was in contact with the ear.

4. Caloric and postrotatory nystagmus were elicitable in these guinea pigs, although modified over that seen in normal animals.

ARGYRIA RESULTING FROM INTRANASAL MEDICATION

A CLINICAL AND EXPERIMENTAL STUDY

BEN L. BRYANT, M.D.
LOS ANGELES

Despite the warnings that have appeared occasionally in the literature, many otolaryngologists still deny the danger of the production of generalized argyria from the use of silver-containing intranasal medication. This heedless attitude is abetted by the advertisements of certain manufacturers of such medications. One of them states quite consistently in the advertising pages of medical publications that the solution is "non-toxic, definitely bacteriostatic, and above all, it is markedly soothing to inflamed tissues."

The physician who has seen even a single victim of full-blown argyrosis, with its typical generalized pigmentation of the skin, giving the patient a bronzed blue or slate color which has been described aptly as the appearance of a corpse suddenly come to life, must necessarily have been impressed with the importance of preventing such a condition. Prevention is actually the only treatment, for, although Stillians [1] and others have shown that by painstaking and multitudinous injections of a 1 per cent solution of potassium ferrocyanide and a 6 per cent solution of sodium thiosulfate in distilled water the skin of the face and neck can be cleared of the pigmentation, a person with the general discoloration due to the distribution of silver will retain it for all of his days.

In the earliest cases reported argyria was caused by the internal use of silver nitrate, but as this form of therapy was relinquished the incidence of argyria naturally diminished, until, within the past generation, the use in the nose and throat of medications containing silver was introduced, with the resultant recrudescence of the lamentable condition.

From the Otolaryngological Research Department of the Good Hope Clinic.

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Read before the Section on Laryngology, Otology and Rhinology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 17, 1939.

1. Stillians, A. W.: Argyria, Arch. Dermat. & Syph. **35**:67-77 (Jan.) 1937.

That argyrosis can and does occur as a direct result of intranasal silver therapy is not denied by those who have had the opportunity to see a large number of patients and who have been alert to perceive the changes in the membranes of the nose and throat and the beginning generalized pigmentation. Some physicians continue to use the dangerous medications because argyria develops comparatively rarely and they feel vaguely that it must be due to some individual physiochemical state. The reason for the apparent disparity between cause and effect was explained by Gaul and Staud.[2] With their biospectrometric analysis (which, incidentally, presents the most convenient and precise method of diagnosis), they have shown that the photochemical action of light produces clinically recognizable argyria when the system contains an amount of silver equal to 8 Gm. of silver arsphenamine. All human beings carry a certain amount of silver with them—in the cells of their bodies—which they have acquired from cooking utensils and cutlery and, in some cases, from the catalytic action between gold and silver dental inlays. If, therapeutically, there is added enough to bring the individual content to the figure mentioned, argyria and its pigmentation are apt to appear.

That the silver which is absorbed is deposited in all the organs of the body has been shown by many workers, among them Blumberg and Carey,[3] Scheffel,[4] Gettler, Gellhorn [5] and Gaul and Staud.[2] It is permanently fixed in the form of small black or grayish granules and has a predilection for connective tissue and elastic fibers. Huet [6] in 1873 and von Kahlden [7] in 1894 reported similar findings in experimental animals which had been given silver nitrate by mouth. Von Kahlden stated that he had seen intracellular silver granules, in contradistinction to many authors, who state that the silver is seldom seen within cells. It is found in the subpapillary layer of the skin and the mucous membranes. Gaul and Staud [2] explained the retention of silver in connective

2. Gaul, L. E., and Staud, A. H.: Clinical Spectroscopy: Study of Biopsy Material from Patients Who Had Received Intravenous Injections of Silver Arsphenamine, Arch. Dermat. & Syph. **30**:433-438 (Sept.) 1934; Clinical Spectroscopy: Seventy Cases of Generalized Argyrosis Following Organic and Colloidal Silver Medication, Including Biospectrometric Analysis of Ten Cases, J. A. M. A. **104**:1387-1390 (April 20) 1935.

3. Blumberg, H., and Carey, T. N.: Argyremia: Detection of Unsuspected and Obscure Argyria by Spectrographic Demonstration of High Blood Silver, J. A. M. A. **103**:1521-1524 (Nov. 17) 1934.

4. Scheffel, C.: The Physiochemistry of Argyria, M. Rec. **140**:205-207, 1934.

5. Gellhorn, G.: Argyria Uteri, Am. J. Obst. & Gynec. **31**:613-618, 1936.

6. Huet, M.: Recherches sur l'argyrie, J. de l'anat. et physiol. **9**:408, 1873.

7. von Kahlden, C.: Ueber die Ablagerung des Silbers in den Nieren, Beitr. z. path. Anat. u. z. allg. Path. **15**:611-625, 1894.

tissue as well as its distribution and excretion by cellular response and the engulfing of the particles by phagocytosis.

Scheffel [4] stated that argyria is caused by the overloading of tissue cells with excessive strengths and added that low concentrations can produce argyria in dehydrated patients or in those with an excessive chloride content in the blood or tissues. Thus the argyria occasionally becoming manifest during pregnancy is an indirect result of vomiting, causing dehydration, which prevents the silver from leaving the cells.

A case representative of several which I have observed and serving to emphasize the situation which results from the ill advised use of silver salts in the nose is that of a 40 year old university professor who when seen first had been treated daily for the preceding three months with a 10 per cent solution of mild protein silver in the form of Dowling packs for the relief of symptoms indicative of chronic ethmoiditis. In addition, he had treated himself on various occasions with nose drops of mild protein silver and cotton applicators soaked with the same substance in the nose. His throat was normal on casual inspection, but viewed with the postnasal mirror the entire nasopharynx looked as though it had been sprayed with soot, and examination of the nose showed that every millimeter of the mucosa was almost black. The only other areas of pigmentation were the finger nails and the conjunctival cul-de-sacs of the lower eyelids. This patient had not had direct instillations of mild protein silver in the eyes except on three widely separated occasions.

Figure 1 is a photomicrograph under high power magnification of tissue removed from the anterior end of the right inferior turbinate of this patient and shows the widespread distribution of silver in the form of grayish black granules in the submucosal tissue, particularly around and in the walls of the blood vessels and in the connective tissue. It is obvious that generalized pigmentation of the skin would have been the fate of this patient, as it has been in many similar instances, had the medication not been discontinued. When it was discontinued and therapy was instituted which permitted the normal physiologic action of the mucosal cilia, his sinal symptoms disappeared. Colloidal silver preparations, as has been demonstrated by Proetz [8] and others, markedly slow the ciliary action by mechanical clogging.

Incidentally and interestingly, after several months the color of the nasal membranes had lightened considerably, indicating that absorption does take place directly from the site of application, despite the rather widespread opinion that the silver is absorbed only from the intestinal tract, after conversion to silver albuminate, and that, therefore, if argyrosis follows intranasal medication it must be because of the solution which is swallowed.

This observation would tend to support the theory of Virchow that reduction and absorption occur at the site of application. This is further

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8. Proetz, A. W.: Treatment of Sinus Disease, Pennsylvania M. J. **39**:385-389, 1936; Nasal Ciliated Epithelium, J. Laryng. & Otol. **49**:557-570, 1934; Effects of Certain Drugs upon Living Ciliated Epithelium, Ann. Otol., Rhin. & Laryng. **43**:450-463, 1934.

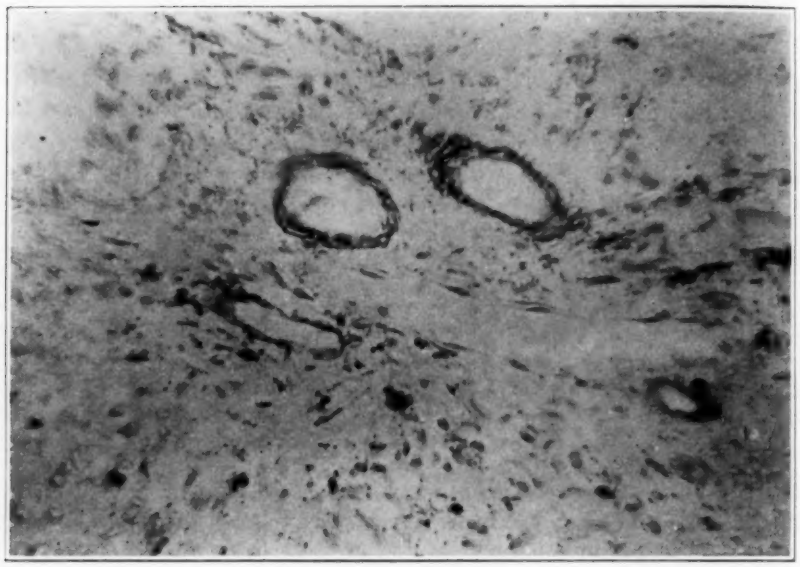

Fig. 1.—High power photomicrograph of tissue removed from the anterior end of the right inferior turbinate of a patient with argyria.

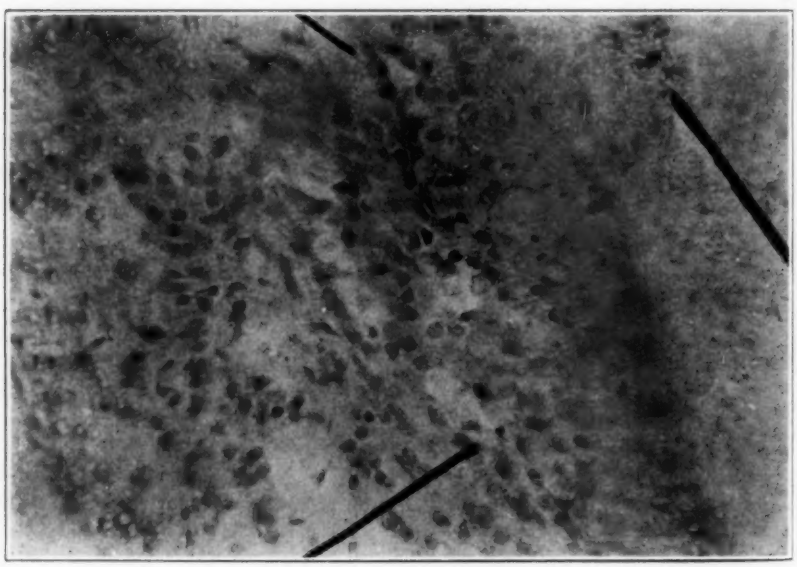

Fig. 2.—High power photomicrograph of tissue from the nasal passages of rabbit 1 (argyrol drops).

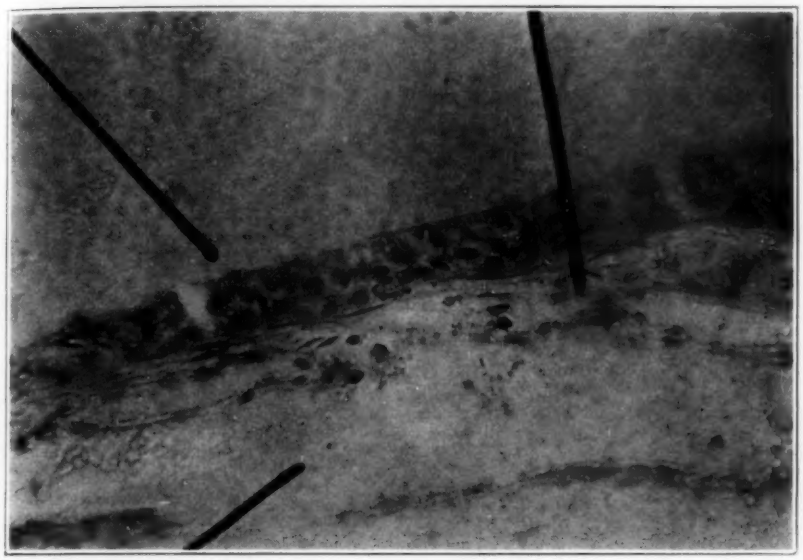

Fig. 3.—High power photomicrograph of tissue from the nasal passages of rabbit 2 (argental drops).

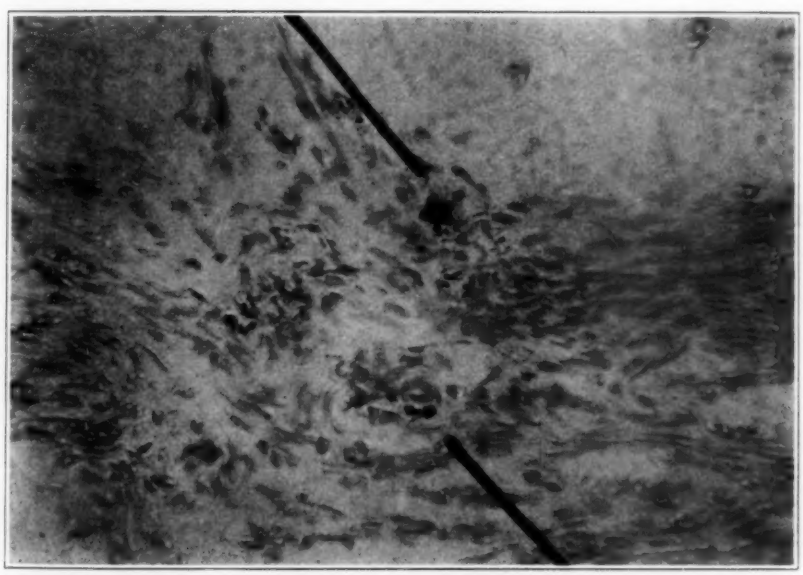

Fig. 4.—High power photomicrograph of tissue from the nasal passages of rabbit 3 (neo-silvol drops).

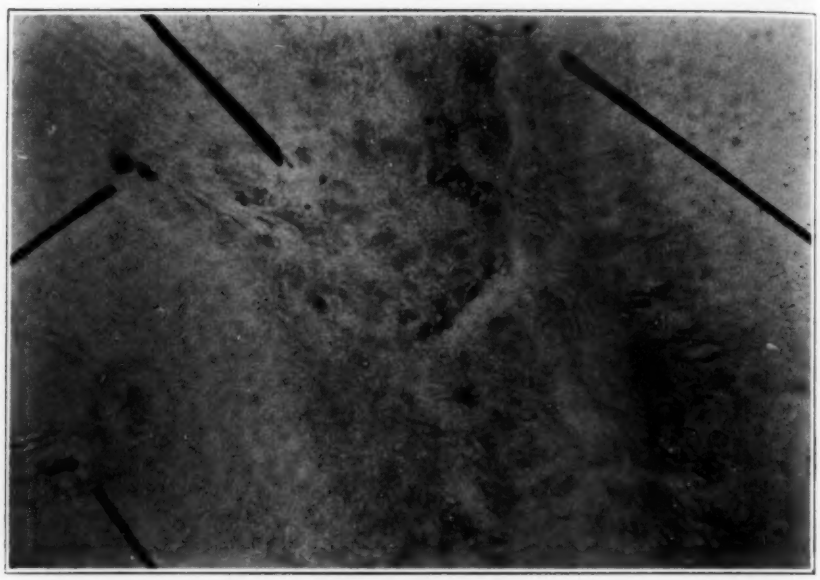

Fig. 5.—High power photomicrograph of tissue from the nasal passages of rabbit 4 (oscol drops).

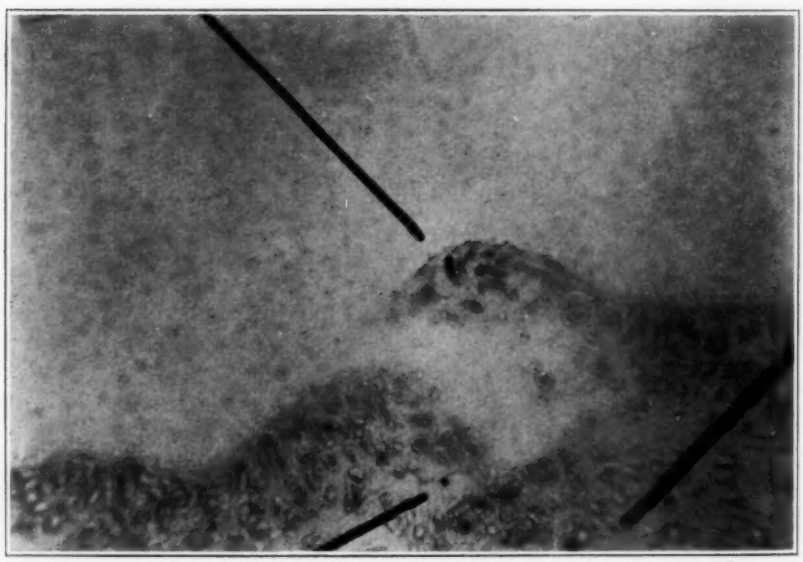

Fig. 6.—High power photomicrograph of tissue from the nasal passages of rabbit 5 (argyrol tampons).

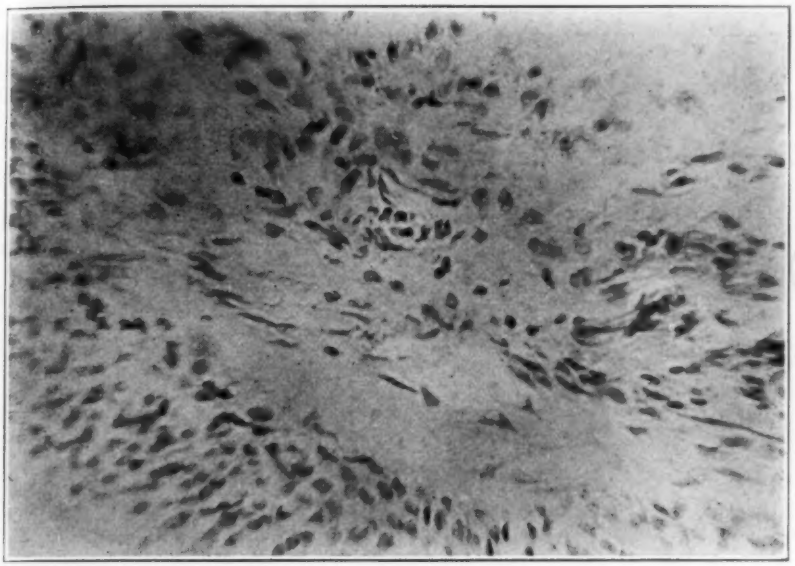

Fig. 7.—High power photomicrograph of tissue from the nasal passages of the control rabbit.

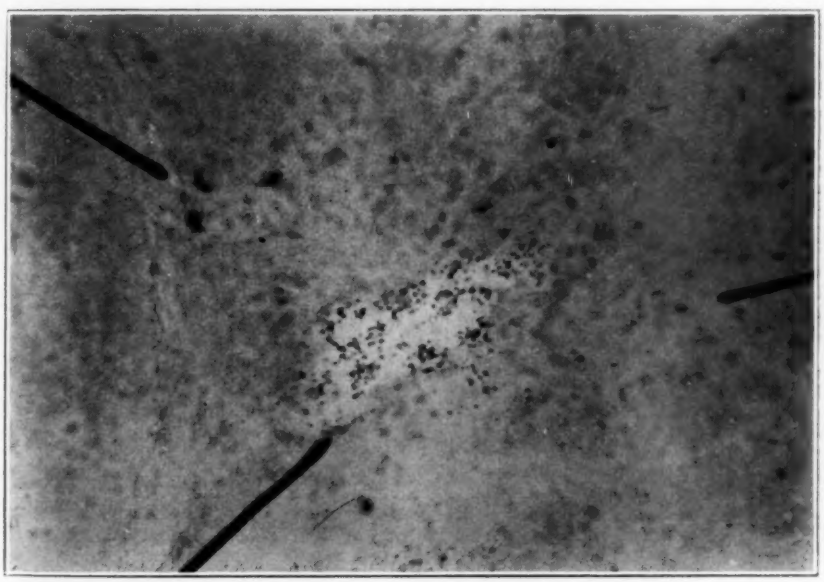

Fig. 8.—High power photomicrograph of tissue from the lungs of rabbit 2.

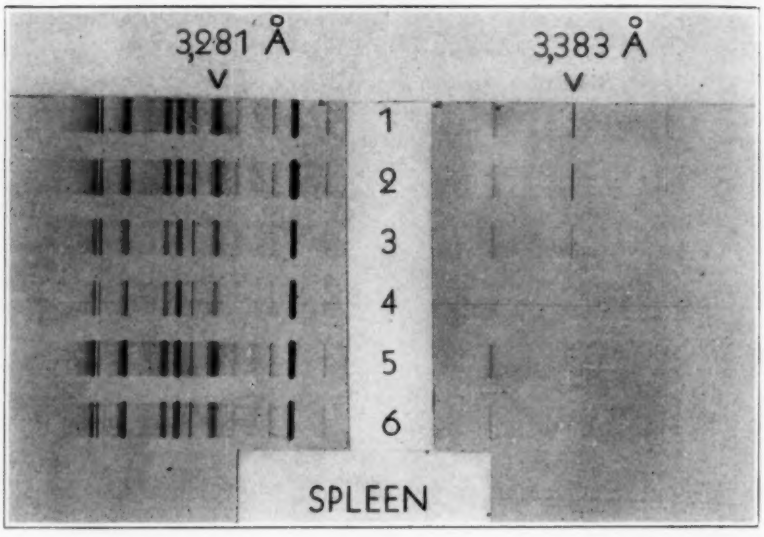

Fig. 9.—Portion of the biospectrographic film of the spleen from the 6 animals.

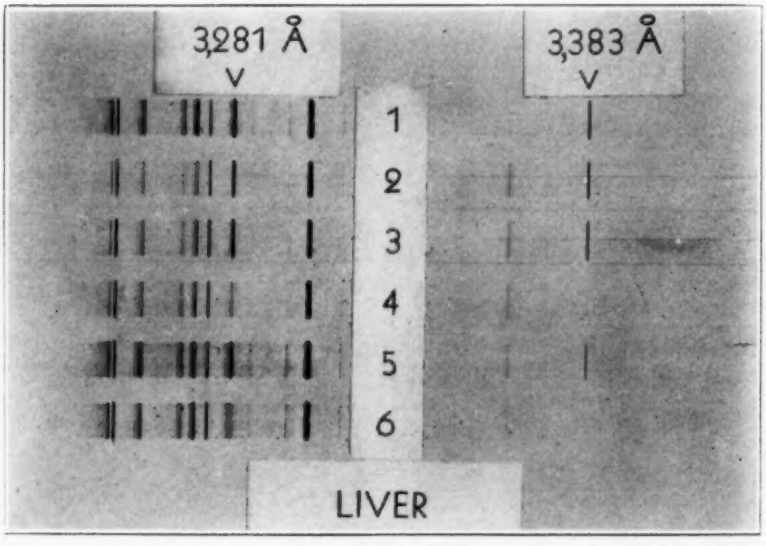

Fig. 10.—Portion of the biospectrographic film of the liver from the 6 animals.

supported by the experimental work of Dworzak, cited by Gelihorn,[5] which showed that when uterine erosions and polyps are treated locally with silver nitrate, impregnation always occurs in two days.

To elucidate this point further and to determine if possible the rate and variation of absorption of different preparations commonly used in the nose, a series of experiments have been carried out on rabbits, the results of which are reported here.

Rabbit 1 had nasal instillations three times daily of a 10 per cent solution of argyrol (a brand of mild protein silver).

Rabbit 2 had nasal instillations three times daily of a 10 per cent solution of argental (a colloidal silver bromide preparation).

Rabbit 3 had nasal instillations three times daily of a 10 per cent solution of neo-silvol (a colloidal silver iodide preparation).

Rabbit 4 had nasal instillations three times daily of a 10 per cent solution of oscol (a colloidal silver preparation).

Summary of Determinations of Silver on Rabbits by Spectrograph

		Content in Milligrams per Hundred Grams of Tissue				
	Animal Number	Spleen, Mg.	Liver, Mg.	Kidney, Mg.	Skin, Mg.	Fur, Mg.
Argyrol, 10%.....	1	0.15	2.0	0.8	0.06	2.3
Argental, 10%.....	2	0.12	1.0	0.12	Not done	0.3
Neo-silvol, 10%.....	3	0.08	2.5	0.05	0.05	1.5
Oscol, 10%.....	4	0.02	0.04	0.02	0.02	0.2
Argyrol, 10%, tampons.....	5	0.02	0.18	0.05	0.02	0.3
Control.....	6	0.02	0.02	0.02	0.02	0.1

Rabbit 5 had cotton tampons of a 10 per cent solution of argyrol (mild protein silver), Dowling packs, in each side of the nose three times a week for two hours at a time.

Rabbit 6 was a control.

At intervals of two, four, six and eight weeks, tissue from the nasal passages of the experimental animals was removed for biopsy. This was sectioned and stained with 1 per cent aqueous solution of safranin. The two week series did not show deposits of silver. The four week series showed a trace in 1 or 2 sections. The six and eight week series showed silver in every specimen examined.

Figures 2 to 6 are photomicrographs of high power magnification of the tissues taken for biopsy. In each of them deposits of silver are visible in the form of grayish black granules or clumps. All of them were made after six weeks' use of the medication, and they are typical of what is seen throughout the slides. Similar observations were made on the sections of tissue made after eight weeks, the only section in which silver did not appear being from rabbit 6, the control.

Figure 7 is a photomicrograph under the same high power magnification of tissue from this control animal.

After six weeks, rabbit 2 (argental drops) died of interstitial pneumonitis, edema and collapse.

Figure 8 is a high power magnification of tissue from the lungs showing scattered deposits of silver in great quantity.

After eight weeks the remaining animals were killed by air embolism, and biospectrometric studies were made to determine the silver content of the liver, kidney, spleen, skin and fur of each animal.

Figure 9 is a portion of the biospectrographic film of the spleen from the 6 animals. A silver line occurs at 3,281 angstrom units, indicated by the caret on the left. It will be noted that band spectrums not of silver appear in many films at 3,280 angstrom units; these can be distinguished from bands representing silver by the sharpness of the silver line. A second silver line appears at 3,383 angstrom units, marked by the caret on the right.

Figure 10 shows the same lines for the liver tissue. It will be noted that in none of these slides is there a measurable silver line for rabbit 6, the control.

Biospectrographic studies of the kidney tissue, the skin and the fur resulted in the same general findings.

A summary of these findings is presented in the table, comparing the various animals and likewise the various tissues examined. Again it will be noted that the control animal has a minimal amount of silver except in the fur, which might well have been contaminated, particularly since the animals after death were inadvertently placed all in one box before being dissected.

COMMENT

Conscientious observers in the field of otolaryngology can no longer doubt the occurrence of generalized argyria caused by intranasal silver medications, especially in view of the 100 per cent increase in such cases reported during a recent five years. A persistent indifferent attitude to this on the part of otolaryngologists might be condoned if it were possible through the use of such medications to achieve beneficial results for the patient which could not be achieved otherwise, but quite the contrary is the case. Hilding, Proetz,[8] Negus and others, through their commendable experimental studies, have brought enlightenment as to the physiology of the nasal mucous membranes, and they have proved the importance of normal ciliary action. The work of Lierle and Moore[9] and of Proetz[8] showed that mild protein silver affects the ciliary beat adversely. In addition, there is an immediate mechanical interference with the highly important streaming of the mucous blanket, caused by the clumping of the drug over the surface. Since the infectious process is beneath the epithelium, it is doubtful that any antiseptic value possessed by colloidal solutions can compensate for their mechanical disadvantages. The same opinion was expressed by Walsh and Cannon[10] as a result of their investigations, which further showed that neither neo-silvol nor argyrol (10 per cent) had any bactericidal effect whatsoever on bronchiectatic pus after forty-four hours, despite the claims of the manufacturers.

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9. Lierle, D. M., and Moore, P. M.: Effects of Drugs on Ciliary Activity of Mucosa of the Upper Respiratory Tract, Arch. Otolaryng. **19**:55-65 (Jan.) 1934.

10. Walsh, T. E., and Cannon, P. R.: The Problem of Intranasal Medication. Ann. Otol., Rhin. & Laryng. **47**:579-607, 1938.

Walsh and Cannon found, moreover, that all their rabbits treated intranasally with 5 per cent argyrol and neo-silvol showed changes in the lungs, such as occurred in rabbit 2, of the present series, consisting of edema, necrosis, desquamation of septal cells and bronchopneumonic lesions. These changes were more marked when the animal in question had the snuffles. This gives one pause to think of the pulmonary lesions occurring especially in infants and children who have had similar nose drops for a preceding cold in the head.

When to these findings is added the real danger of the production of argyrosis, it would seem that a clear case against the use of the various silver preparations intranasally had been established and that they should be supplanted generally by the use of weak ephedrine or neosynephrin in an isotonic solution as a vehicle, since these have been proved when so used to have no deleterious effect on the lung tissue or on the action of the cilia and at the same time to achieve the only possible beneficial result, i. e., vasoconstriction and relief of congestion. It seems to be a duty also to disprove the advertised statements that "the argyrol tampon and the argyrol spray have become standard practice," and that there is "perfect safety in the use of 25-50 per cent applications and 10-25 per cent solutions at home." There has come to my attention only one preparation, namely, solargentum (a brand of mild protein silver), which carries on its label the following warning: "Caution: The prolonged use of any silver preparation may produce irremediable discoloration of the skin or mucous membrane (argyria)." Such printed warnings should be made mandatory to discourage self medication with silver salts over protracted periods, since this practice too often is the forerunner of argyria.

SUMMARY AND CONCLUSIONS

The generalized pigmentation of argyria is permanent; therefore, its only reasonable treatment is prevention.

Generalized argyria is apt to result from the photochemical action of light when the body carries silver equal to 8 Gm. of silver arsphenamine.

Silver in the usual intranasal medication is absorbed by the nasal membranes and distributed throughout the body.

Absorption of silver is experimentally demonstrable after six weeks of regular medication and in some instances after four weeks. In this study, its presence was proved by microscopic examination of the nasal tissues and by biospectrometric examination of several of the internal organs.

Widespread generalized deposits of silver were demonstrated throughout the tissue of the lungs of an experimental animal which

died from bronchopneumonic lesions as the result of the use of intranasal medication containing silver.

The use of silver-containing nasal medication over even a few weeks is dangerous and accomplishes nothing that cannot be accomplished safely and more efficiently by other means.

Measures should be taken to see that the public is warned of the danger of the production of argyria by long-continued self medication with silver preparations. All such preparations intended for use on mucous membranes should carry a warning statement on their labels.

Dr. Roy W. Hammack, pathologist, sectioned, stained and examined the tissues. Dr. Albert L. Chaney, biochemist, carried out the biospectrographic studies. Mr. Russell Stimson, of the firm of Heimann, Monroe and Jones, made the photographs.

2007 Wilshire Boulevard.

ABSTRACT OF DISCUSSION

Dr. Gordon F. Harkness, Davenport, Iowa: The internal administration of silver nitrate, though formerly the greatest cause of argyria, has been relegated to a minor place through the discontinuance of such medication.

My bibliography of sixty-five articles since 1931 reveals an even greater danger from the local application of true silver colloids or of silver salts in combination with protective colloids. Many families include these silver preparations in the "family medical cabinet" but are unaware of the dire consequences from prolonged self medication.

For a long time in our practice my associates and I have had "nonrefillable" on every such prescription.

To epitomize, a certain concentration of silver, largely intercellular, together with exposure to light, natural or artificial, seems essential to the condition recognized as argyria of the skin. That it can occur by direct absorption through mucous membranes has been established. The absorption of silver nitrate or lactate can be understood. The absorption of colloidal medicaments through local applications brings one into the realm of colloidal chemistry, which is beyond the ken of most otolaryngologists. While the physical overloading of the tissues is an important link, certain not well understood chemical alterations are essential to the clinical picture. This applies first to the absorption of colloids through animal membranes. The splitting of free silver ions at the site of application and their distribution in a soluble form, followed by the intercellular deposition in the form of an insoluble silver salt, may take place long before the clinical recognition of the condition.

Biospectroscopic examination will reveal the amount of silver retained in the tissues before any objective signs are apparent.

Clinical proof of argyria without clinical symptoms has recently come to my attention.

A woman now 47 received in 1927 one hundred capsules of silver nitrate or some silver-containing compound for so-called stomach trouble. Since that time she has not received medication with silver. Not until 1932, five years after medication, was the bluish gray tinge of her skin first noted. Since that time it has been constant and progressive. This, to my mind, clinically substantiates the presence of argyria of the tissues for a long time without clinical manifestations.

Many people who have argyria of the tissues for a long time without clinical manifestations are potential candidates for the recognizable type of the condition.

It seems that the insoluble silver salt most likely to produce this condition is silver chloride.

Whether it is the excess of chlorides in the body fluids, the p_H of body tissues, an idiosyncrasy or a sensitivity that precipitates the condition, no one has satisfactorily explained.

Silver chloride, being white, can be deposited without clinical evidence. Its oxidation, after it has become sensitive to light, with color changes, is a slow but progressive process.

Effect of treatment, except in small areas, is practically nil.

Until more is definitely known of the chemical alterations, the relief of the condition lies in the prevention, the avoidance of the indiscriminate use of silver preparations, the therapeutic efficacy of which has been largely exaggerated.

INTERSTITIAL EMPHYSEMA OF THE LUNG WITH SPONTANEOUS PNEUMOTHORAX AND SUBCUTANEOUS EMPHYSEMA

DEMONSTRATION OF AIR IN THE SEPTUMS OF A HUMAN LUNG

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AND
MORRIS E. STERN, M.D.
NEW YORK

Pneumothorax with subcutaneous and mediastinal emphysema has long been known to occur in association with increased intrapulmonary pressure. Obstructive lesions of the larynx, trachea and bronchi are not infrequently accompanied by these conditions, which may become evident after tracheotomy.

Mediastinal and subcutaneous emphysema, with or without pneumothorax, may occur also in otherwise healthy persons who do not present any evidence of increased intrapulmonary pressure.

These conditions, occurring singly or simultaneously, have been recently brought to the attention of the members of the medical profession by Hamman,[1] McGuire and Bean [2] and Graebner.[3]

The pathogenesis of pneumothorax with mediastinal and subcutaneous emphysema was never explained satisfactorily with the aid of postmortem examinations, as no definite rupture of the lung and parietal pleura was demonstrated in the majority of necropsies. The pathogenesis of these conditions was, therefore, until recently a matter of conjecture.

Bullowa [4] described subcutaneous emphysema without pneumothorax occurring in association with epidemic influenza. He suggested that

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From the Diphtheria Service and from the Pathologic Laboratories of the Willard Parker Hospital.

Presented in part at a meeting of the Section of Otolaryngology of the New York Academy of Medicine, April 19, 1939.

1. Hamman, L.: Spontaneous Mediastinal Emphysema, Bull. Johns Hopkins Hosp. **64**:1, 1939.

2. McGuire, J., and Bean, W. B.: Spontaneous Interstitial Emphysema of the Lung, Am. J. M. Sc. **197**:502, 1939.

3. Graebner, H.: Pneumopericardium and Pneumomediastinum in Cases of Acute Obstructive Laryngitis, Arch. Otolaryng. **29**:446 (March) 1939.

4. Bullowa, J. G. M.: Tissue Emphysema in Influenza, M. Rec. **95**:346, 1919.

rupture of the alveoli in those parts of the lung which were free from pneumonia and were overdistended by inspired air was responsible for the subcutaneous emphysema.

Macklin [5] presented an experimental explanation of the events leading to mediastinal emphysema, pneumothorax and subcutaneous emphysema in overdistention of the lungs. By overinflating the lungs of cats until pneumothorax developed, he succeeded in showing that the air from the distended alveoli entered the potential spaces of the perivascular sheaths in the interlobular septums. From these perivascular spaces the air traveled along the blood vessels to the mediastinum, spreading from there to the mediastinal pleura, to the subcutaneous tissues of the neck and to the retroperitoneal tissues. Minute ruptures in the blebs of the mediastinal pleura resulted in pneumothorax. Macklin could demonstrate this route of the air by filling the bronchial tree of the collapsed lung with fixing fluid until the lung was redistended to approximately the normal volume. This opened not only the collapsed alveoli but the septal spaces containing air. Microscopic sections from lungs treated in this manner showed air in the perivascular sheaths.

Graebner,[3] in reporting 5 cases of mediastinal emphysema associated with obstructive laryngitis, stated the opinion that escape of the air into the mediastinum takes place by the route suggested by Macklin.

Hamman,[1] in describing cases of mediastinal emphysema in normal adults, some of whom had associated pneumothorax, also accepted Macklin's explanation of the mechanism.

While sections from lungs of experimental animals showed the spread of air from the alveoli into the septum, this mechanism of pneumothorax has not been demonstrated in histologic sections from human lungs.

A case of diphtheria with obstruction of the respiratory tubes by membrane observed at this hospital afforded, apparently, the first opportunity to observe this mechanism in man.

REPORT OF A CASE

W. B., a white boy aged 2½ years, was admitted to the Willard Parker Hospital on Dec. 26, 1938, at 7 p. m. The child had been transferred from another hospital in the city.

Past History.—The child had undergone tracheotomy for a foreign body in the larynx in July 1937, at the age of 1 year. After the operation he had bronchopneumonia and empyema due to Streptococcus haemolyticus. The latter condition cleared up with intercostal drainage.

In July 1938 the child again had empyema due to Str. haemolyticus and Staphylococcus albus. He recovered after a thoracotomy.

5. Macklin, C. C.: Pneumothorax with Massive Collapse from Experimental Local Over-Inflation of Lung Substance, Canad. M. A. J. **36**:414, 1937.

There was a questionable history of injections of toxoid. No Schick test had been performed. There was no history of contagious diseases.

Present Illness.—This began six days before the child's admission to the hospital from which he was transferred, when he had an infection of the upper respiratory tract, was lethargic and had a temperature of 103 F. He improved until the day of admission, when he became drowsy, vomited twice, was prostrated and had labored respirations. The temperature was 99.6 F., and cyanosis was noted. Culture of material taken from the throat three days before admission failed to reveal Klebs-Loeffler bacilli.

On admission to one of the hospitals in New York city on December 25, the patient was cyanotic. The respirations were rapid, with marked retractions. The temperature was 101.8 F.; the respiratory rate, 70 and the pulse rate, 130. While material for culture was being taken from the throat, the patient ceased to breathe; an emergency tracheotomy through the old tracheotomy scar was performed, and the child was resuscitated. Six thousand units of diphtheria antitoxin was given intramuscularly on admission. Culture of material from the throat gave negative results for diphtheria bacilli. On the following day 12,000 units of diphtheria antitoxin was given, one half of which was given intravenously and the rest intramuscularly. At one time during this day the patient became cyanotic, and a plug of membrane was removed from the tracheotomy tube. A culture of material from this yielded diphtheria bacilli, and he was transferred to the Willard Parker Hospital, to the service of Dr. Arthur S. Wilson.

On admission, on December 26, the child was moribund. He appeared very pale, with marked cyanosis of the nail beds; slight suprasternal retractions were evident. Emergency treatment was instituted immediately. The tracheotomy tube was removed; a laryngoscope was introduced, and suction of the larynx and trachea was done with a metal tracheal catheter. This procedure resulted in the removal of a large amount of thick white-gray membrane, including a tracheal cast. The larger bronchi were also treated by suction. The patient was given stimulants, oxygen by catheter and dextrose intravenously. These measures gave relief. Further examination at this time revealed a white-gray membrane on both tonsils, the uvula and the posterior pharyngeal wall. Cultures of material from these membranes showed the presence of virulent Klebs-Loeffler bacilli. Examination of the heart revealed sounds of poor quality, with a rapid rate, no arrhythmia and no murmurs. Examination of the lungs showed slight dulness over the left side of the chest anteriorly and posteriorly. The breath sounds came through fairly well over the right side of the chest but were diminished over the left. The liver was palpable 5 cm. below the costal margin. The temperature was 101.6 F., the respiratory rate 66 and the pulse rate 110. A diagnosis of laryngotracheobronchial diphtheria involving the tonsils, uvula and pharynx was made.

Further treatment at this time consisted in administration of 40,000 units of diphtheria antitoxin, one half of which was given intravenously and the rest intramuscularly. The tracheotomy tube was reinserted, and the child was given symptomatic and supportive treatment.

On the following day, at 9 a. m., the child appeared somewhat improved, but still acutely ill. Examination of the lungs revealed the breath sounds to be coming through well in both pulmonary fields. The color was good. The respiratory rate was 58, and the temperature, 101 F. The heart sounds were improved; no murmurs were heard, and the rate was 156. The liver was still enlarged. The membrane was still present on the tonsils, uvula and pharyngeal wall. Roentgen examination of the lungs at this time revealed no abnormality. No pneumothorax was present.

At 11:45 a. m. on December 28 the child began to have labored respirations, and suction at the bedside with a soft rubber catheter through the tracheotomy tube was of no avail. A laryngoscope was then introduced, and the larynx, trachea and bronchi were aspirated, with removal of many large pieces of white-gray membrane. Relief followed immediately. Examination of the lungs after this procedure revealed the breath sounds to be coming through well in both fields.

At 6:50 p. m. the same day the patient became very pale and ceased to breathe. Artificial respiration, stimulants, oxygen and intravenous injections of dextrose were given, and the patient was resuscitated. Aspiration was again performed, with removal of large pieces of membrane.

At 8:45 p. m. the child again became very pale; labored respirations were evident. Subcutaneous emphysema of the left side of the neck and the left anterior part of the wall of the chest was noted. Examination of the lungs at this time revealed a mediastinal shift to the right, with hyperresonance and diminished breath sounds on the left side. A diagnosis of pneumothorax on the left side was made. Aspiration of the larynx, trachea and bronchi was of no avail. A needle was placed in the left pleural space; a catheter attached to the hub of the needle was placed under water, and a large amount of air was drawn off. Owing to the extreme restlessness of the patient and the difficulty of maintaining the needle in place, this treatment was discontinued. The child had two mild convulsions and became more dyspneic and restless. He died at 11:48 p. m.

Necropsy.—Necropsy was performed eleven hours after death. The body was well developed and well nourished. A low tracheotomy incision, 2 cm. long, ending 1 cm. above the suprasternal notch, was present in the midline. The edges of the wound were thick and fibrotic. There was subcutaneous emphysema of the left side of the neck and of the left lateral abdominal wall subcostally. An old thoracotomy scar was present in the seventh intercostal space, in the right posterior axillary line.

The necropsy incision was limited to a downward prolongation for 5 cm. of the tracheotomy wound.

The edges of the wound were firmly adherent to the underlying tissues; the thyroid gland was divided during the tracheotomy.

Water was poured into the lower angle of the cutaneous incision, and both pleurae were punctured under water. Puncture of the left pleura resulted in a considerable escape of air bubbles; no air escaped from the right pleura.

The mediastinum could not be visualized, owing to the limitations of the incision. When the thoracic organs were removed, no blebs of air were seen over the thymus.

The right lung was entirely adherent to the parietal pleura over all its aspects; it was fairly well aerated except for several firm areas suggestive of early pneumonia.

The left upper lobe was partially adherent to the chest wall anteriorly and posteriorly; the lower lobe was adherent posteriorly. The adhesions were moderately strong, and apparently not recent. Parts of the lung were atelectatic, while several areas were firm and apparently bronchopneumonic. The rest of the lung was air holding. Edematous fluid appeared from the cut surface of the lung on pressure. The bronchioles contained a mucoid exudate.

The mucosa of the larger bronchi and of the trachea was rough, but no membrane was present. Some shreds of membrane were adherent to the congested laryngeal mucosa. A sheet of old scar tissue extended from the upper end of the tracheotomy wound to the vocal cords, without causing obstruction. The tonsils could not be reached through the low incision.

No air blebs were present over the pericardium. The parietal pericardium was entirely adherent to the visceral leaflet. Pleuropericardial adhesions were present on both sides. The heart weighed 95 Gm.

The liver and spleen were slightly enlarged and anemic. The adrenal glands were normal. The kidneys were slightly congested.

Microscopic Examination of the Lungs: Areas of emphysema, of edema, of intra-alveolar hemorrhage and of bronchopneumonia were present in both lungs.

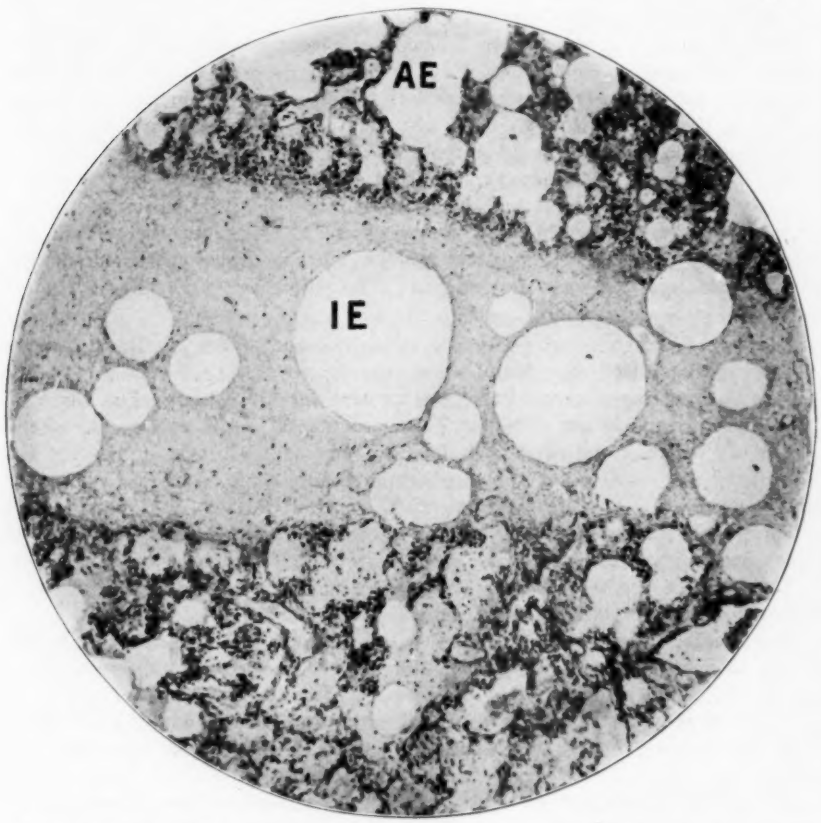

Fig. 1.—Air in the interlobular septum, giving it a "Swiss cheese" appearance. The septum is wide and edematous. *AE*, alveolar emphysema; *IE*, interstitial emphysema. Hematoxylin and eosin. × 50.

Large parts of the left lung showed areas of atelectasis. Sections of two blocks of tissue from the left lung showed wide, edematous interlobular septums, with small hemorrhages. Large, round empty spaces, apparently bubbles of air which had escaped from the adjacent alveoli, were present in the septums, giving them a "Swiss cheese" appearance (fig. 1). A number of empty spaces were lined with flat cells resembling endothelium. In a section from one of the blocks some of the air spaces were partly filled with coagulated protein (fig. 2). The round spaces containing air were therefore identified as distended lymphatics.

Many small bronchi were collapsed. Some of the medium-sized bronchi showed congestion of the wall and desquamation of the epithelium and contained shreds of fibrinous membrane. No air-holding spaces were seen in the interstitial tissue in sections from the blocks taken at the root of the lung.

Toxic Klebs-Loeffler bacilli were recovered from the culture of heart's blood taken post mortem. It was impossible to determine whether the bacteremia was present during life or whether the bacilli entered the heart's blood from the adjacent organs after death.

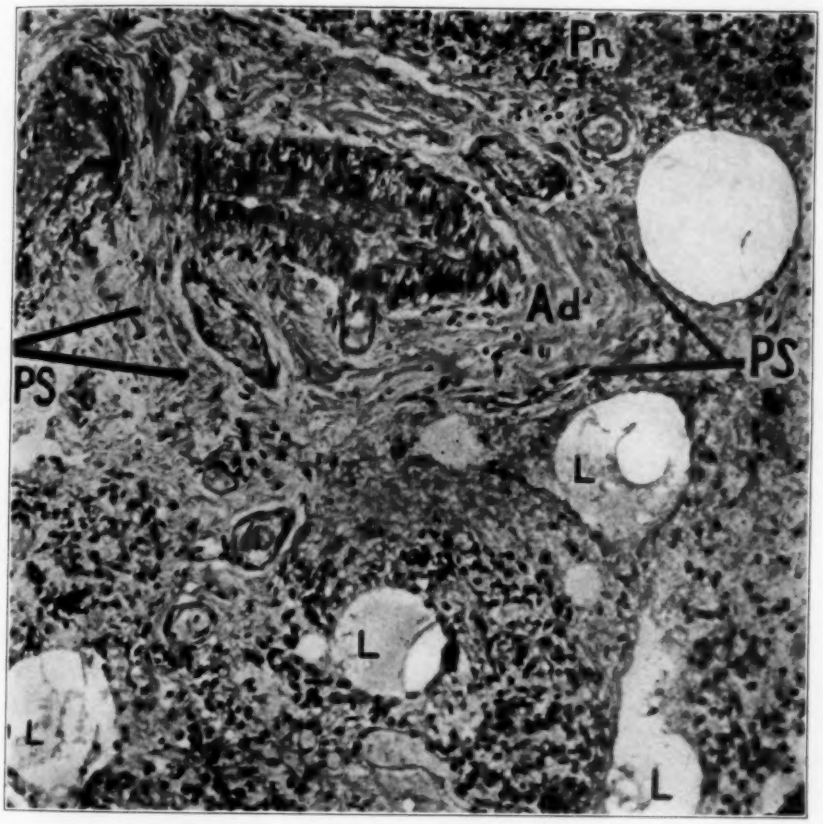

Fig. 2.—Location of air outside the perivascular sheaths. *PS,* perivascular sheath; *Ad,* adventitia; *L,* lymphatics, some of which are partly filled with air; *Pn,* pneumonia. Hematoxylin and eosin. × 170.

COMMENT

The case presented was one of interstitial emphysema of the lung in a patient with diphtheria complicated by spontaneous pneumothorax and subcutaneous emphysema.

To our knowledge, no other cases are on record in which interstitial emphysema of the lung has been demonstrated in histologic sections of human lungs in cases of spontaneous pneumothorax. The demonstration

of air in the septums in histologic preparations was possible only because the lung did not fully collapse after the pneumothorax occurred, being held in partial expansion by pleural adhesions and by bronchopneumonia. The partial expansion kept open the alveoli not affected by pneumonia and the septal spaces containing air. These conditions closely approximated those produced by Macklin by reexpansion of the collapsed lungs of cats.

As there was no mediastinal emphysema in this case, the derivation of the subcutaneous emphysema and of the pneumothorax from the interstitial emphysema of the lung may require a somewhat different

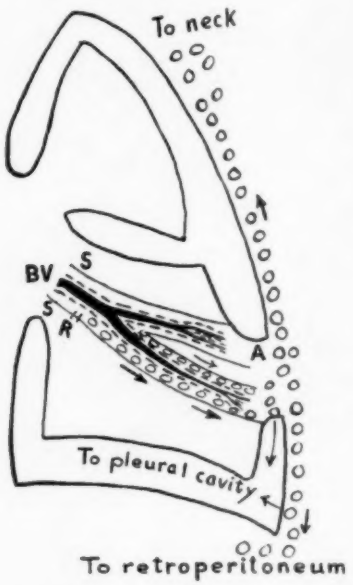

Fig. 3.—Diagram illustrating the mechanism of pneumothorax and subcutaneous emphysema in the presence of pleural adhesions and in the absence of mediastinal emphysema. *S-S*, septum; *BV*, blood vessel; dotted line, potential perivascular sheath; *A*, adhesion; *R*, alveolar rupture.

explanation from that given by Macklin. In our case the air in the septums was seen not in the perivascular sheaths but in septal lymphatics outside these sheaths. It is probable that from the septal lymphatics the air traveled not to the hilus but to the periphery of the lung, i. e., to the lymph channels of the septal adhesions, and escaped from there into the pleural cavity and into the retropleural connective tissue. The air could also have found its way into the pleura through the rupture of retropleural blebs (fig. 3). From the retropleural tissues the air could readily have spread into the subcutaneous tissues of the neck and the

abdomen. This short route, from ruptured alveoli through pleural adhesions into the subcutaneous tissue, has been mentioned by several authors (von Bergmann;[6] Neffson and Bullowa[7]).

The rupture of the alveoli probably occurred in a part of the lung which was free from bronchopneumonia and which had been overinflated because of severe and prolonged interference with normal respiration by the diphtheritic membrane and because of vicarious emphysema.

The tracheotomy apparently had no bearing either on the pneumothorax or on the subcutaneous emphysema, as it was performed through an old tracheotomy scar and through the thyroid gland, which would hardly have permitted any aspiration of air into the surrounding tissues; besides, the pneumothorax occurred three days after the tracheotomy.

SUMMARY

A case is reported in which a patient with obstruction of the respiratory tubes by a diphtheritic membrane, not relieved by tracheotomy and late administration of antitoxin, had unilateral pneumothorax and subcutaneous emphysema without mediastinal emphysema. As the lung was not fully collapsed, because of bronchopneumonia and old pleural adhesions, microscopic sections revealed the interstitial emphysema of the septums, with air in the distended lymphatics.

The air in the septums probably traveled to the periphery of the lung and escaped through the pleural adhesions into the pleural cavity and into the subcutaneous tissue.

Dr. Arthur S. Wilson gave us permission to report this case.

6. von Bergmann, G.: Das mediastinale Emphysem, in von Bergmann, G., and Staehelin, R.: Handbuch der inneren Medizin, Berlin, Julius Springer, 1928, vol. 2, pt. 1, p. 669.

7. Neffson, A. H., and Bullowa, J. G. M.: Influenza with Simultaneous Bilateral Spontaneous Pneumothorax and Subcutaneous Emphysema: Report of a Case, with Comment on the Mechanism of Production, Arch. Otolaryng. **28**:388 (Sept.) 1938.

BENIGN TUMORS OF BRONCHI

WITH SPECIAL REFERENCE TO VASCULAR ADENOMA

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In the rapidly accumulating literature on benign bronchial tumors, one type stands out as deserving special consideration. Variously designated as "bronchial adenoma," "adenomatous polyp" and "vascular adenoma," it is a type of growth which can now be fairly accurately identified histologically and recognized clinically. Being the most common, it deserves to be considered apart from the motley group of benign endobronchial tumors which have been thrown together under one heading in the literature.

Any bronchial growth if left untreated ultimately produces occlusion of a bronchus with resulting atelectasis, drowned lung and pulmonary suppuration. Earlier recognition, which is now occurring with increasing frequency, permits treatment before irreparable damage to the lungs has been done. Of the bronchial new growths the benign vascular adenomas appear to show the most constant clinical course. Study of the case histories in the literature bears this out.

Since publication of the opinion of Jackson and Jackson,[1] most of the benign bronchial tumors have been considered due to stagnation of purulent secretion and have been designated as "inflammatory tumors." Peroni,[2] on the basis of experimental work, likened them to hyperplastic and degenerative processes in the nose and larynx and preferred to designate them as "inflammatory polypi."

According to others, vascular adenomas appear to possess the characteristics of real blastomas. It is difficult to believe that they are primarily inflammatory in origin, because they are seldom found in cases of long-standing bronchiectasis and pulmonary abscess.

These tumors may exist for years without producing signs of obstruction, but by their tendency to cause a characteristic type of hemoptysis

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Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

1. Jackson, C., and Jackson, C. L.: Benign Tumor of the Trachea and Bronchi, with Special Reference to Tumor-Like Formation of Inflammatory Origin, J. A. M. A. **99**:1747 (Nov. 19) 1932.

2. Peroni, A.: Inflammatory Tumors of Bronchi, Arch. Otolaryng. **19**:1 (Jan.) 1934.

they frequently may be diagnosed before the lung has been severely damaged. That this is true is shown by the experience of Wessler and Rabin,[3] who were able to make a correct diagnosis in 9 of 14 cases.

Pollak, Cohen and Gnassi [4] up to 1937 collected 104 recorded cases of primary benign tumor or inflammatory hyperplastic tumor-like formation of the bronchial tree, diagnosed at autopsy or by bronchoscopic biopsy. Almost half of these (51) were adenomas. Twenty-three, or 22 per cent, were included under "inflammatory hyperplastic growths"; among these were such formations as simple polyps, papillomas and granulomas. The rest formed a mixed group, consisting of fibromas, chondromas, lipomas, leiomyomas and lymphomas.

PATHOLOGIC DATA

Adenomas may represent a congenital anomaly (Fried [5]). This author stated that they originate from the cells lining the bronchial mucous glands; Wessler and Rabin expressed the belief that they more nearly resemble the cells lining the ducts of these glands. Fried affirmed that mucous glands are not found in bronchi less than 10 mm. in diameter. Adenomas are, accordingly, limited to the walls of the larger bronchi—an important fact which is borne out clinically.

Mallory [6] has advanced the opinion that histologically they more nearly resemble carcinoid tumors of the intestinal tract. He contended that, although of slow growth, they sometimes show a tendency to invade the bronchial wall and cannot be classified as exclusively benign. Womack and Graham [7] have recently contributed a new point of view, likening them to mixed tumors of the parotid gland, including their potentiality for malignant change. In addition to bronchoscopic observations they had the opportunity of studying the pathologic character of these tumors after lobectomy and pneumonectomy. They saw in them a resemblance to fetal lung tissue and stressed their many points of similarity to mixed tumors. This study is important because, as the investigators pointed out, few adenomas have been discovered at autopsy. Womack and Graham stated that many undergo malignant

3. Wessler, H., and Rabin, C. B.: Benign Tumor of Bronchus, Am. J. M. Sc. **183**:164 (Feb.) 1932.

4. Pollak, B. S.; Cohen, S., and Gnassi, A. M.: Inflammatory Bronchial Tumors: Report of a Case and Review of the Literature, Arch. Otolaryng. **27**:426 (April) 1938.

5. Fried, B. M.: Adenoma of Bronchial Mucous Gland, Arch. Otolaryng. **20**:375 (Sept.) 1934.

6. Adenoma of the Bronchus, Cabot Case 24091, New England J. Med. **218**:390 (March 3) 1938.

7. Womack, N. A., and Graham, E. A.: Mixed Tumors of the Lung: So-Called Bronchial and Pulmonary Adenoma, Arch. Path. **26**:165 (July) 1938.

change, which makes impossible their postmortem differentiation from ordinary far advanced bronchogenic carcinoma. This represents a reversion to the belief held some years ago, when many adenomas were considered by pathologists to be adenocarcinoma. As Jackson and Konzelmann [8] pointed out, pathologists who have previously diagnosed certain growths as carcinomas now admit doubt as to the malignancy of the tumors, in view of survival of the patients for many years.

These tumors occur most commonly in women between 30 and 40 years of age; carcinomas are found four times as frequently in men between the ages of 40 and 60.

Benign tumors of the bronchi grow slowly and usually first encroach on the bronchial lumen in the form of a polypoid elevation beneath the mucosa. Later they become elongated and pyriform, their free ends projecting upward toward the trachea, this direction being imparted to them by the accompanying irritating cough. Many become pedunculated.

The body of the tumor is usually covered by a layer of bronchial epithelium which frequently shows squamous metaplasia. The epithelium rests on an intact basement membrane under which is a layer of loose connective tissue containing an abundant supply of dilated blood vessels. This is such a prominent finding that recently the tumors have been referred to as "vascular adenomas." [9] The main bodies of such tumors are composed of polyhedral cells with large round or oval reticular nuclei arranged either in acini or in solid columns. The cells rest on a definite basement membrane, are uniform and homogeneous in structure and staining qualities and usually lack mitotic figures. However, at times the cells extend deep to the cartilage plates of the bronchus, indicating the difficulties of completely eradicating the growth. The presence of abundant capillaries between the groups of tumor cells speaks against malignancy. Because the cells are not always arranged in glandlike formation (fig. 1) the term "adenoma" is unacceptable to some.

BRONCHOSCOPIC APPEARANCE

Through the bronchoscope an adenoma appears as a rather smooth, rounded, congested, cherry-like growth, frequently with blood vessels coursing underneath its epithelial covering. There is freedom of movement to the end of the bronchoscope, which is often not present in

8. Jackson, C. L., and Konzelmann, F. W.: Bronchoscopic Aspect of Bronchial Tumors, with Special Reference to So-Called Bronchial Adenoma, J. Thoracic Surg. **6**:312 (Feb.) 1937.

9. Zamora, A. M., and Schuster, N. H.: Vascular Adenoma of Bronchus, J. Laryng. & Otol. **52**:337 (May) 1937.

malignant disease. Slight contact with the end of the bronchoscope or the suction tube is apt to release a brisk hemorrhage.

SYMPTOMS AND DIAGNOSIS

Clinically there is a typical latent period, during which cough may be absent, but hemoptysis in this stage is common and deserves separate consideration. Morlock and Pinchin[10] cited the case of a patient in whom hemoptysis was the sole symptom for over twenty years. In some women it may occur only during the menstrual period. It differs from

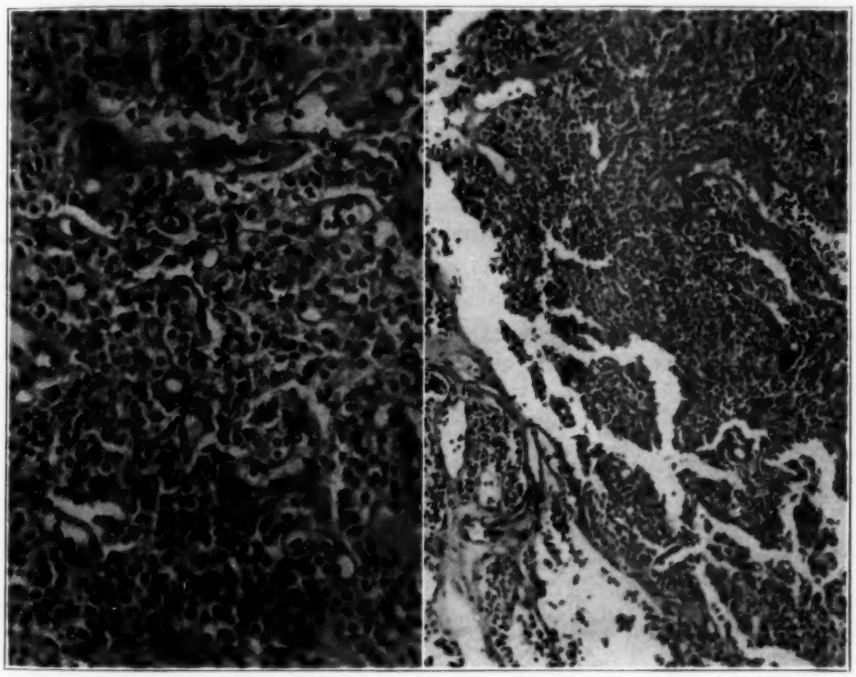

Fig. 1.—Low and high power photomicrographs of a specimen of tissue from a benign vascular adenoma.

the bleeding of parenchymal lesions of the lung in that it occurs suddenly, without warning, and may cease just as abruptly without appreciable further streaking. Coming from a large bronchus, its downward course impeded by the tumor mass, the blood is expectorated before it can collect in the smaller bronchi. Turner,[11] on the other hand, sug-

10. Morlock, H. V., and Pinchin, A. J. S.: Benign Neoplasms of Bronchus, with Records of Nine Cases, Brit. M. J. **2**:332 (Aug. 24) 1935.

11. Turner, P. A.: Benign Tumors of the Bronchus, with Especial Reference to Early Diagnosis: Two Case Reports, Kentucky M. J. **31**:423 (Sept.) 1933.

gested that these growths predispose to bronchospasm in the presence of sudden hemorrhage, thus preventing aspiration of the blood and explaining the sudden cessation of bleeding. He based his supposition on the fact that iodized poppyseed oil would not enter below the point of attachment of the tumor even in a case in which the lumen of the bronchus was not obliterated by the growth. In 1 of my patients iodized oil did fill the bronchi, outlining a few sacculations (figs. 2*B* and 2*C*).

There is frequently a history of preceding attacks of pneumonia, and it is fair to assume that many were attacks of pneumonitis following atelectasis from bronchostenosis. Because of their marked vascularity, adenomas are prone to undergo changes in size and shape, thus producing succeeding attacks of atelectasis. Ultimately, if they are unrecognized and untreated, drowned lung, bronchiectasis and pulmonary suppuration ensue. Infection has been known to spread to the pleura, producing effusion and empyema.

Soreness in the affected side of the chest is a common complaint.

The so-called asthmatoid wheeze, an important sign of bronchial obstruction, which is best heard at the open mouth, may frequently be elicitated. In many cases the condition has been confused with bronchial asthma because of the attacks of paroxysmal wheezing. When the tumors have long pedicles, patients may have extreme attacks of dyspnea on suddenly changing their position. When hemoptysis predominates, the condition is frequently mistakenly diagnosed as pulmonary tuberculosis.

In the early stages physical findings and roentgen evidence may be lacking. However, when the tumor has attained a size sufficient to occlude a bronchus and interfere with ventilation and drainage, symptoms and signs of atelectasis and pulmonary suppuration will soon become apparent.

ROENTGEN FINDINGS

Two roentgen signs which hitherto have not been stressed may be helpful in diagnosis. They cannot replace bronchoscopic observations as the final arbiter, but they may be used to advantage in stressing to both patient and physician the need for this examination. One deals with the changes found in "lung mapping" and the other with the so-called triangular shadow at the base of the lung. Careful study of films taken after filling of the bronchi with iodized poppyseed oil may reveal the outline of the tumor mass (figs. 2*B*, 2*C*, 3*A* and 3*B*) and may also indicate the lack of filling of the tributary portion of lung (fig. 3). The second sign, the "triangular shadow at the base of the lung," is sometimes demonstrable in the presence of atelectasis of a lower lobe. This finding, of course, obtains only when the tumor obstructs the entrance to a lower lobe. Experience has shown that this is actually the most common site of these growths. Rosenblum and

Klein [12] have referred to this recently in reporting the case of a boy 11 years old in whom they found an adenomatous polyp in the right main bronchus, producing atelectasis and bronchiectasis of the right lower lobe. In 1 of my cases, when this triangular shadow was noted on the roentgenogram it was at first thought to be a distortion of the mediastinum. Since I became acquainted with the work of Anspach,[13] who showed that in children it is produced by an atelectatic lower lobe and is a precursor of bronchiectasis, its interpretation has become clearer. In none of the 50 children studied by Anspach was the atelectasis caused by an obstructing bronchial tumor; in the majority of instances it was due to infection and in 3 cases to a foreign body. *A* and *B* of figure 2 clearly show this triangular basal shadow as distinct from the right border of the heart.

TREATMENT

In comparison to what is known of the pathologic and clinical aspects, information regarding the exact management of these tumors is meager. The physician encountering his first case is often at sea as to the best type of procedure.

From a study of the reported cases it is found that five types of treatment are available: (1) removal through the bronchoscope; (2) electrocoagulation; (3) radium treatment; (4) roentgen treatment, and (5) partial or total pneumonectomy.

Bronchoscopic Removal.—With such vascular tumors as adenomas this appears to be a difficult and dangerous procedure. Physicians who have observed the release of an alarming flow of blood by mere contact of the suction tube with the tumor have no great inclination to use biting forceps. Although usually the hemorrhage may be controlled by suction and by sponging with epinephrine, sufficient blood may have reached the pulmonary parenchyma to produce alarming postoperative pneumonitis (case 1).

Artificial pneumothorax instituted and maintained prior to instrumentation may obviate this danger. I adopted this measure in 1 instance, with gratifying results. Subsequent treatment after failure to maintain the pneumothorax (because of the patient's lack of cooperation) again produced postoperative reaction. The method was used with the idea that compression of the lung would prevent blood from reaching the alveoli, thus minimizing the incidence of parenchymal infections. The use of pneumothorax for benign tumors was sug-

12. Rosenblum, P., and Klein, R. I.: Adenomatous Polyp of Right Main Bronchus Producing Atelectasis, J. Pediat. **7**:791 (Dec.) 1935.

13. Anspach, W. E.: Atelectasis and Bronchiectasis in Children: Study of Fifty Cases Presenting Triangular Shadow of Base of Lung, Am. J. Dis. Child. **47**:1011 (May) 1934.

gested to me by a recommendation made by Pressman and Emery[14] in the treatment of bronchial carcinomas. These authors stated that they have found it extremely valuable prior to the intrabronchial application of a basket containing radium needles.

Electrocoagulation or Surgical Diathermy.—This measure has recently been stressed by Kernan,[15] who has contributed some important details. It should be tried only with the region under local anesthesia, an attempt being made to preserve the cough reflex. Kernan has designed a special bronchoscope with a separate channel for the active electrode, which allows for better vision. He has expressed his preference for a bead-shaped rather than a pointed tip on account of the risk of penetrating the bronchial wall. Surgical diathermy is apt to cause a greater shrinking of the growth than is seen immediately after a treatment, because the vitality of the tissue away from the point of contact is also affected. The treatment should not be too strenuous, because of the danger of scarring and stenosis. This was impressed on me by observation of the end result in case 4. In this case, after various types of treatment at another clinic during the past ten years, the tumor had apparently receded but the orifice of the right main bronchus was left no larger than a pinhead.

Removing the major portion of the growth by the use of a biting forceps and following this immediately by coagulation is also dangerous, on account of the risk of penetration. Coagulation treatments may be repeated every two weeks. Secondary hemorrhage as late as two weeks after coagulation is possible. It occurred in 1 of my cases in an alarming manner.

Radium Treatment.—This may be applied by direct implantation of radon seeds into the tumor mass or by placing adjacent to it a tube containing seeds or needles.

Radon seeds of gold or platinum may be inserted and left in place for the desired number of days, or they may be implanted with the idea of allowing them to remain permanently. When so placed they seemingly find firm anchorage and remain embedded. In case 3 radon seeds were implanted ten years prior to the time of writing and are still to be observed roentgenographically at the site of the tumor. One cannot help wondering what deleterious foreign body effects would result if the seeds were lost in the smaller bronchioles.

Belsey,[6] citing the plan adopted at the Brompton Hospital, London, England, stated that twelve seeds, each containing 1.25 millicuries, are distributed within the jacket of a canalized tubular container, which is

14. Pressman, J. J., and Emery, C. K.: New Method of Radium Application in Cancer of Bronchus, Ann. Otol., Rhin. & Laryng. **46**:314 (June) 1937.

15. Kernan, J. D.: Electrocoagulation in Bronchogenic Conditions, Arch. Phys. Therapy **19**:38 (Jan.) 1938.

placed alongside the tumor through the bronchoscope and maintained in position by a spring for seven days and then removed. Pressman and Emery have adopted a similar plan for treating bronchial carcinoma, inserting a "bird cage" containing radium needles around the growth. The cage is left in place from thirty to sixty-five hours.

High Voltage Roentgen Therapy.—This method has been used in some of the cases cited in the literature, but in most of them it appears to have been utilized only as an adjunct to other forms of treatment.

Surgical Extirpation.—Removal of the affected lobe or lung by lobectomy or pneumonectomy is destined to find increasing favor, especially in cases in which the tumors have been long unrecognized and untreated and irreparable pulmonary damage has been done. With the advances in pulmonary surgery this form of treatment may also play an increasingly popular role even in cases in which the pathologic process is not so far advanced.

REPORT OF CASES

CASE 1.—R. B., a woman aged 30, was admitted to St. Joseph's Hospital May 18, 1937, under the care of Dr. G. H. Curfman. She complained of cough, bloody expectoration, wheezing and pain in the right side of her chest. She had been told by one physician that she had tuberculosis and by another that she had asthma. She had had two attacks of pneumonia in childhood. For the past six years she had been short of breath. A roentgenogram of the lungs showed increased peribronchial shadows and a triangular area of density at the base of the right lung (fig. 2*A*). The presumptive diagnoses were asthmatic bronchitis and bronchiectasis. I was asked to instil iodized poppyseed oil for "lung mapping." On questioning of the patient the possibility of bronchial tumor suggested itself and I advised bronchoscopic examination.

Bronchoscopic Examination.—A tumor mass the size of a navy bean was revealed. It was red and vascular, with a sessile attachment to the medial bronchial wall; it nearly obstructed the entrance to the right lower lobe bronchus. When the growth was touched with the suction tube a profuse hemorrhage occurred. Biopsy specimens were taken, and 10 cc. of iodized popyseed oil was instilled into the right main bronchus. Histologic examination showed a vascular polypoid growth with no evidence of malignancy.

Bronchoscopic Treatment (June 30).—Attempts were made to encircle the tumor with a cold wire snare and also with a diathermy loop, without success. Several pieces were removed with a punch forceps. This was followed by electrocoagulation. Bleeding was brisk. Histologic examination of the material removed at this sitting showed "large, distended vascular channels." The diagnosis was angioma of the bronchial mucosa. On the third day there were a rise in temperature to 104 F. and signs of consolidation in the lower lobe of the right lung. On July 15 the patient had a severe hemorrhage.

Artificial Pneumothorax.—Pneumothorax was decided on in order to collapse the right lung prior to the next bronchoscopic treatment, in an attempt to minimize the dangers from hemorrhage. This was done and another bronchoscopic treatment was given on Aug. 7, 1937, at which time the tumor was again coagulated. Little reaction followed, and the patient left the hospital on the fifth day.

On September 2 electrocoagulation was repeated. The lung had been allowed to reexpand because the patient had refused further refills. On the following day there was a rise in temperature to 102 F., which lasted for two weeks. A roentgenogram of the chest showed an area of density at the base of the right lung, possibly with fluid. The patient finally recovered but was advised to have radon implantations at a later bronchoscopic treatment in view of the severe bleeding and aspiration pneumonia which had followed the preceding treatments.

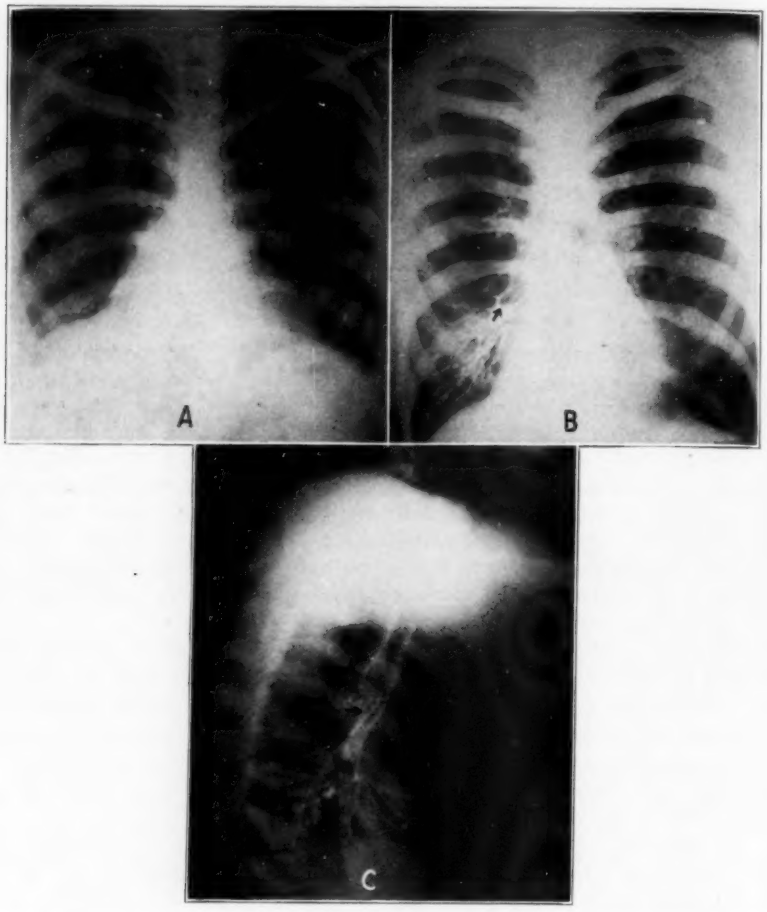

Fig. 2 (case 1).—*A*, roentgenogram of the chest, showing a triangular shadow at the base of the right lung. The right border of the heart is distinctly seen and is separate from this shadow, which indicates atelectasis of the lower lobe. *B*, roentgenogram taken after filling of the bronchi with iodized poppyseed oil, showing the outline of the tumor and the triangular shadow. The arrow points to the probable outline of the tumor. *C*, lateral roentgenogram taken after injection of iodized oil, showing the outline of the tumor and a few sacculations in the lower lobe of the right lung, indicative of early bronchiectasis. The arrow points to the probable outline of the tumor.

CASE 2.—R. H., a man aged 29, first entered St. Joseph's Hospital under the care of Dr. C. A. Shepard on Oct. 19, 1935, with a diagnosis of pneumonitis of the lower lobe of the left lung. He recovered, but during the succeeding year he continued to raise varying quantities of bright red blood at intervals. Physical and roentgen examinations repeatedly gave negative results. Two bronchoscopic examinations had been made by another bronchoscopist, but no definite tumor mass or bleeding area was seen. After an unusually severe hemorrhage, pneumothorax was induced and was maintained for several months.

Bronchoscopic Examination.—On Feb. 4, 1938, a large mass was revealed in the left main bronchus, close to the orifice of the branch to the lower lobe. The tumor extended along the posterolateral wall of the left main bronchus, filling more than half of its lumen. A moderate amount of bleeding followed the removal of a biopsy specimen, which was reported to show "angioma of the bronchial wall."

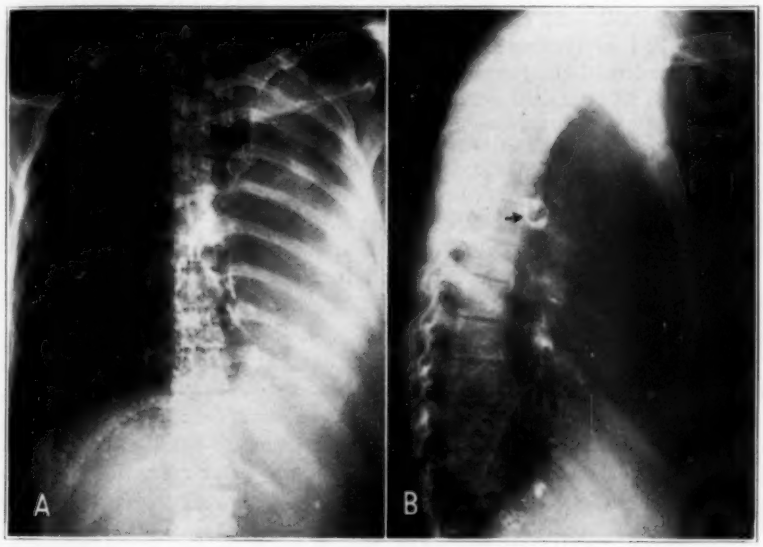

Fig. 3 (case 3).—*A*, roentgenogram of the chest after filling of the bronchi with iodized poppyseed oil, showing the outline of the tumor in the region of the orifice of the left main bronchus. The left lung is atelectatic, and the trachea is pulled over to the left. *B*, lateral roentgenogram taken after filling of the bronchi with iodized oil, showing the outline of the tumor (indicated by arrow) at the bifurcation of the trachea.

Electrocoagulation was then applied. On March 7 three radon seeds were implanted in the tumor mass. A roentgenogram of the chest taken several weeks later showed the radon seeds in place. The patient then left the city, apparently in good health. In a letter seven months later he reported an increase in the severity of his symptoms. He had had numerous bronchoscopic treatments without improvement. He added that he would soon undergo a lobectomy, which had been recommended in an eastern clinic.

CASE 3.—A G., a woman aged 42, entered the Denver General Hospital Feb. 20, 1937. She had been in the ward for patients with tuberculosis for the past

two months. She had suffered repeated sudden hemorrhages during the past four years. The roentgen rays revealed atelectasis of the left lung, the trachea being markedly deviated to the left. After filling with iodized poppyseed oil, the outline of a tumor mass blocking the entrance to the left main bronchus could be distinctly made out. The iodized oil did not enter the left lung (fig. 3).

Bronchoscopic Examination.—On March 18 Dr. C. H. Darrow made a bronchoscopic examination, which showed a lobulated tumor mass 1.5 to 2 cm. in diameter, located at the carina and filling the left main bronchus. The tumor extended slightly into the right main bronchus and bled easily. Apparently it was not pedunculated. The report on the biopsy specimen was "low grade adenocarcinoma." This was followed by a course of roentgen treatments of a calculated dosage of 1½ erythema skin doses. A roentgenogram taken April 30 was thought to show about one-third reduction in the size of the tumor. On May 6, three gold radon seeds containing 6 millicuries were implanted in the tumor mass. On May 21, two additional radon seeds were inserted. When last heard from the patient had had no further hemorrhages.

Reexamination of the biopsy specimen revealed the structure and arrangement of the cells to be typical of nonmalignant vascular adenoma (fig. 1).

CASE 4.—E. S., a woman aged 37, came to the outpatient department of the National Jewish Hospital with the following history: After an attack of pneumonia in 1927 she was left with a dull, aching pain in the right side of her chest and an irritating cough. There was no bleeding. In March 1930 a smooth tumor almost occluding the right main bronchus and attached to the right side of the trachea just above the level of the bifurcation was found at a bronchoscopic examination made in New York city. The report of the biopsy specimen was "adenomatous hyperplasia of mucous glands, possibly carcinoma."

In September 1936 a diagnosis of adenoma (cylindroma) of the bronchus was made. The patient stated that radon seeds had been implanted ten years previously. Repeated cauterizations had been done until June 1937, followed by a course of twelve roentgen treatments. A roentgenogram of the chest taken on Oct. 20, 1938 showed two radon seeds in the region of the orifice of the right main bronchus.

Bronchoscopic Examination.—On April 15 the lumen of the right main bronchus was shown to be narrowed to the size of a pinhead. This was dilated with Jackson bougies ranging in caliber up to no. 18 F. The patient reported definite improvement for about four months, but at the time of writing she has noticed an increase in wheezing and shortness of breath for one month.

SUMMARY AND CONCLUSIONS

Three cases of benign bronchial tumor are added to the literature. A fourth case, in which the patient was treated elsewhere and is now under my care on account of fibrotic bronchial stenosis, is also cited.

The majority of benign bronchial tumors fall into the category of benign adenoma, which should be regarded as an entity. Although the histologic diagnosis is now better established, pathologists still disagree on many important points, including the degree of malignancy of such a tumor. The history of a characteristic type of hemoptysis, often

over a period of years, together with the physical signs and bronchoscopic findings, should receive equal consideration with biopsy reports in diagnosing this type of growth.

Bronchographic examination may help in outlining the tumor, but it should never replace accurate bronchoscopic examination.

When the growth obstructs a lower lobe, the demonstration of a triangular shadow at the base of the lung, indicating atelectasis, may afford a clue by pointing to the need for a diagnostic bronchoscopic procedure.

Artificial pneumothorax should be considered prior to bronchoscopic removal or destruction by electrocoagulation because of its value in reducing the incidence of posthemorrhagic aspiration pneumonia.

Treatment by bronchoscopic methods has resulted in many apparent cures or arrests and should be tried first. When this is unsuccessful, lobectomy and pneumonectomy offer an increasingly favorable outlook.

Case 3 is reviewed by permission of Dr. C. H. Darrow.

DIPLACUSIS: A LOCALIZING SYMPTOM OF DISEASE OF THE ORGAN OF CORTI

THEORETIC CONSIDERATIONS, CLINICAL OBSERVATIONS AND PRACTICAL APPLICATION

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The finding of diplacusis in a case of deafness is a valuable diagnostic aid in localizing the pathologic condition, since diplacusis can be caused only by a lesion of the organ of Corti.

This statement is based on theoretic considerations, which I shall discuss, and on clinical observations, which I shall describe. If it is true, some practical applications follow for the diagnosis and treatment of certain cases of deafness and of Ménière's syndrome.

INTRODUCTORY OBSERVATIONS

Definition.—Diplacusis is the hearing of one sound as two. There are three varieties of diplacusis: Diplacusis binauralis echotica, in which a sound is heard a fraction of a second later by one ear than by the other, as a distinct echo, is comparatively rare. I have observed it but once (case 8). Diplacusis monauralis dysharmonica, in which a pure tone is heard by the affected ear as a double tone of two different pitches, also is a rare symptom which I have seen but once (case 4). Diplacusis binauralis dysharmonica, in which the same sound is heard at a different pitch by the two ears, is rather frequent in aural disease. It is this type which is referred to generally in the literature, and unless otherwise specified in this paper, as diplacusis.

Testing for Diplacusis.—Disharmonic diplacusis is more common than one would infer from the literature. Musicians in whom diplacusis develops are usually annoyed by it and complain of it. The great majority of patients with diplacusis, however, are not aware of it until they are specifically tested for it. The ordinary hearing tests, including the audiogram, will not elicit the symptom. Diplacusis must be specifically tested for.

The test for diplacusis is made with tuning forks, and a complete series by octaves, from 64 to 2048 vibrations should be employed, since diplacusis rarely affects the entire tonal scale to an equal degree and

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may be limited to a part of it. In testing for diplacusis it is of the utmost importance that the patient hear the fork with equal loudness in each ear, for otherwise many patients will confuse loudness with pitch. The vibrating tuning fork is held alternately in front of each ear, the distance being regulated so that the patient hears the tone equally loud in the two ears. He is asked whether he hears the same tone in the two ears. When true diplacusis is present the patient states that he hears an entirely different sound in one ear from that which he hears in the other, the pitch being definitely higher or lower. With this difference in pitch, may occur an alteration in the quality of the sound also, so that it is rasping, harsh, unpleasant or even painful to the diseased ear as contrasted to the pleasant musical tone heard by the normal ear. It is helpful to have the patient hum the note that he hears in each ear. An occasional patient who is unmusical will be unable to distinguish pitch but will still recognize that the quality of the sound is entirely different in his two ears.

Theoretic Considerations.—Many diverse explanations of diplacusis have been offered. Because it is sometimes seen in cases of otitis media, many authors, including Politzer, Gradenigo and Jacobsen, expressed the belief that alterations in the sound-conducting apparatus accounted for diplacusis in at least some cases. In 1920 Schilling [1] made the first positive statement that diplacusis is always due to structural alterations in the vibrating elements of Corti's organ. In 1923 Shambaugh and Knudsen,[2] unaware of Schilling's publication, reached the same conclusion on the basis of a clinical study of 10 cases of diplacusis, stating that the alteration is such that a segment of the membrane which normally vibrates to a certain tone subsequently vibrates to a tone of a higher or lower pitch. In 1935 I [3] described a syndrome consisting of low tone nerve deafness, diplacusis and attacks of vertigo. I pointed out that on theoretic considerations and in view of recent work on the physiology of hearing which substantiated the theory of selective localization of tone in the cochlea, diplacusis could only be caused by a distortion of the vibrating membrane in the organ of Corti and could not be caused by any lesion in the conducting mechanism, the eighth nerve or the central acoustic tracts (Fig. 1).

In 1937 Proetz [4] experienced temporary diplacusis in his own ear. From his observations in his own case he concluded that diplacusis is due to a lesion of the vibrating membrane in the organ of Corti.

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1. Schilling, R.: Ueber musikalisches Falschhören, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **105**:65, 1920.

2. Shambaugh, G. E., and Knudsen, V. O.: Report of an Investigation of Ten Cases of Diplacusis, Tr. Am. Otol. Soc. **16**:397, 1923.

3. Shambaugh, G. E., Jr.: Syndrome of Diplacusis and Nerve Deafness for Low Tones, Arch. Otolaryng. **21**:694 (June) 1935.

4. Proetz, A. W.: Diplacusis Binauralis Dysharmonica, Ann. Otol., Rhin. & Laryng. **46**:119, 1937.

In the absence of pathologic proof, which up to the present time is lacking, reliance must be placed on clinical evidence to prove or disprove the theoretic statement that diplacusis is due to a lesion of the organ of Corti.

CLINICAL OBSERVATIONS

The clinical observations are based on a series of 45 patients with diplacusis observed during the past few years. These patients were all seen in routine office practice in which the tuning fork tests for diplacusis are included as part of the routine hearing tests. The 45 patients with diplacusis had the following aural conditions: Thirty-three had primary nerve deafness; 5 had suppurative otitis media, 3 acute and 2 chronic; 3 had had a Lempert operation for fistulization of the labyrinth for otosclerosis; 2 had otosclerosis; 1 had catarrhal otitis media, and 1 had otherwise normal hearing.

OTHERWISE NORMAL HEARING WITH DIPLACUSIS (ONE CASE)

CASE 1.—Mrs. R. L. D., aged 41, complained of roaring in the right ear for five months and hoarseness off and on for several years. The tuning forks were

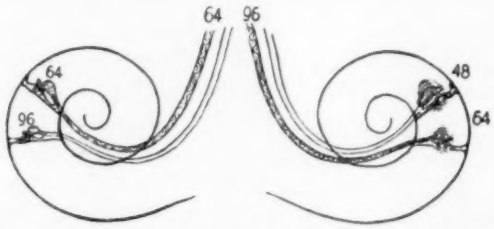

Fig. 1.—In the normal ear the note of a fork of 64 double vibrations stimulates its special portion of the organ of Corti and its set of nerve fibers. In the affected ear the vibrating membrane has been altered so that the area which previously responded to the fork of 96 double vibrations now responds to that of 64 double vibrations, the tone of the latter fork being heard in the brain as one of 96 double vibrations, the result being diplacusis.

heard equally well in both ears but slightly higher pitched and with an uncomfortable reverberation in the right ear. The right vocal cord was edematous in the middle third. The basal metabolic rate was —18 per cent. On administration of thyroid the tinnitus and diplacusis improved, and they had nearly disappeared when the patient moved away, six weeks later.

It is possible that in this case the diplacusis was due to edema of the vibrating membrane in the organ of Corti caused by thyroid deficiency. The patient is the only person I have seen with diplacusis and otherwise normal hearing.

CATARRHAL OTITIS MEDIA WITH DIPLACUSIS (ONE CASE)

CASE 2.—Mr. R. E. P., aged 61, complained of fulness and slight deafness in the right ear for two days, and his wife's voice sounded unpleasantly harsh in that

ear. Hearing tests showed a slight conduction defect in the right ear, chiefly for the 32 and 64 vibration forks, with diplacusis, these forks being heard at a higher pitch and sounding less musical in that ear. Inflation of the eustachian tube with a catheter relieved the fulness and improved the hearing but did not help the diplacusis, which did not clear up until several days later.

In this case the diplacusis was evidently not due to the tubal occlusion, since it was not relieved by inflation. There was probably coincident edema of the vibrating membrane in the cochlea.

OTOSCLEROSIS WITH DIPLACUSIS (TWO CASES)

CASE 3.—Mrs. L. Mc. M., aged 39, complained of progressive deafness for eighteen years. Her father had had progressive deafness. The drum membranes were normal, with a distinct pink glow from the right promontory. Hearing tests revealed a bilateral conduction defect, with diplacusis for the 2048 vibration fork, which was heard one and a half tones higher in the left ear than in the right. The

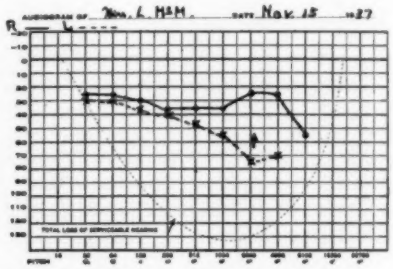

Fig 2 (case 3).—Otosclerosis with primary fixation of the stapes in both ears and secondary degeneration of the nerve in the left ear (evidenced by the loss of hearing for high tones); diplacusis for the 2048 frequency fork, which is heard 1½ tones higher pitched in the left ear.

diagnosis was otosclerosis with primary fixation of the stapes and secondary labyrinthine involvement on the left side as evidenced by the loss of hearing for high tones and the diplacusis (fig. 2).

CASE 4.—Mr. A. K. O., aged 35, was first seen by my father sixteen years ago, because of gradual impairment of hearing. Bilateral high tone nerve deafness was found, but because of a family history of deafness and a distinct pink glow from the promontories the diagnosis of probable primary labyrinthine otosclerosis was made. The nerve deafness slowly increased until eighteen months before the time of this report, when the loss of hearing was accelerated and the tuning fork tests began to show a conduction defect in addition to the nerve deafness, indicating beginning ankylosis of the stapes. At the time of writing, diplacusis has been present for nine years for the 2048 vibration fork, which is heard at a higher pitch in the right ear. A month ago monaural diplacusis for the 1024 vibration fork developed in the left ear, so that this fork is heard as a double tone in this one ear. The diagnosis is primary labyrinthine otosclerosis with secondary fixation of the stapes (fig. 3).

Diplacusis is unusual in otosclerosis. Both the patients clearly had labyrinthine involvement for the high tones, which the diplacusis affected, so that the diplacusis could be accounted for by a lesion of the vibrating membrane in the cochlea.

POSTOPERATIVE DIPLACUSIS (THREE CASES)

CASE 5.—Mrs. B. P., aged 45, complained of progressive deafness for four years. The drum membranes were normal, and hearing tests showed a conduction defect. The Lempert fistulization for otosclerosis was carried out in July 1938. The operation was uneventful, with marked improvement in hearing on the table. The first day after operation there was second degree nystagmus, and the second day there was severe vertigo, nausea and vomiting. The tuning forks were heard in both ears, but diplacusis was present for the 512 to 4096 vibration forks, which were all heard at a higher pitch in the ear operated on. Two days later the diplacusis had spread to include also the 128 and 256 vibration forks. One month later the diplacusis was still present, but there was no reaction to the fistula test nor caloric response from the horizontal canal, and the hearing was not improved over the preoperative level.

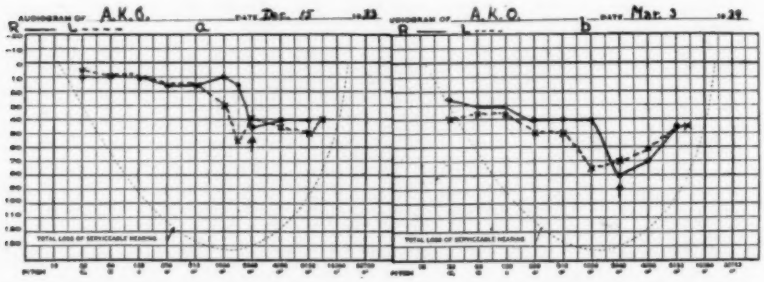

Fig. 3 (case 4).—Otosclerosis with primary nerve deafness (*a*), secondary fixation of the stapes developing fifteen years after onset (*b*); diplacusis for the 2048 frequency fork, which is heard at a higher pitch in the right ear.

This was clearly a case of postoperative serous labyrinthitis resulting in loss of function of the horizontal canal and involving the cochlea, as evidenced by the diplacusis, but not producing a dead labyrinth.

CASE 6.—Miss M. C., aged 28, complained of progressive deafness for twelve years. The drum membranes were normal, and the hearing tests were characteristic of conduction deafness. The Lempert fistulization for otosclerosis was carried out in July 1938. The membranous horizontal canal was ruptured, as evidenced by the empty appearance of the lumen when the bony canal was opened. There was immediate marked improvement in hearing, which lasted forty-eight hours without vertigo or nystagmus. Diplacusis for the two highest-pitched forks, 1024 and 2048 vibrations, was present on the second postoperative day, and a week later it had spread to involve all the forks, so that they were heard at a higher pitch in the ear operated on. At the time of writing the ruptured membranous canal has healed, as evidenced by a strongly positive reaction to the fistula test, which is active nine months after operation. The diplacusis has diminished until

only the 256 and 2048 vibration forks are heard at a slightly lower pitch in the ear operated on. The hearing is only slightly improved over the preoperative level (fig. 4).

It is significant that when the membranous labyrinth was ruptured diplacusis first developed for the tones which produce vibration in the part of the organ of Corti closest to the operative trauma, that is, for the high tones, and gradually spread to involve the low tones. The patient, incidentally, has absolute pitch, and she was able to report accurately the exact difference in pitch between the two ears.

Case 7.—Mr. P. L. J., aged 41, complained of progressive deafness for fifteen years. The drum membranes were normal, and the hearing tests showed a conduction defect. The Lempert operation for otosclerosis was carried out in October 1938. The membranous horizontal canal was accidentally ruptured, as evidenced by the empty lumen of the bony canal and failure to respond to the fistula test on the table. A week later, however, the membranous canal had healed, and there was an active response to the fistula test, which is still present at the

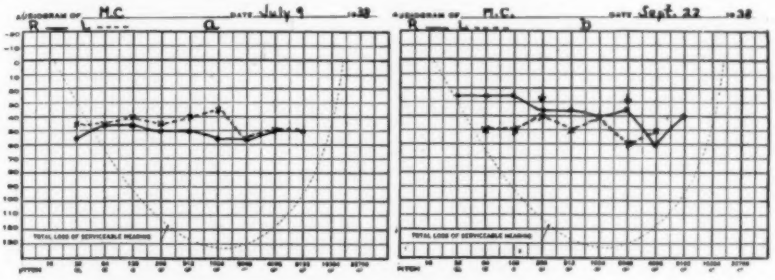

Fig. 4 (case 6).—Preoperative hearing in a case of otosclerosis with primary fixation of the stapes (*a*); hearing after fistulization of the labyrinth (right ear), diplacusis for the 256 and 2048 frequency forks, which are heard at a lower pitch in the ear operated on (*b*).

time of writing, seven months later. Diplacusis first appeared, nine days after operation, for the 1024 vibration fork, and two days later, for all the forks, which were heard at a higher pitch in the ear operated on. The diplacusis cleared up two months later. The hearing was not improved at any time over the preoperative level.

In 5 more patients with otosclerosis operated on by the Lempert procedure, the membranous labyrinth was not injured, and there was no postoperative serous labyrinthitis. In none of these 5 patients did diplacusis develop, indicating that diplacusis after the Lempert operation means a lesion of the membranous labyrinth involving the cochlea.

SUPPURATIVE OTITIS MEDIA WITH DIPLACUSIS (FIVE CASES)

Case 8.—Miss C. F., aged 42, complained of severe earache on the left for two days, with vertigo, nausea, vomiting and ataxia for one day. She was found

to have acute suppurative otitis media with spontaneous nystagmus toward the affected ear, indicating serous labyrinthitis. Diplacusis for all the forks was present, all the forks being heard at a higher pitch in the affected ear. The otitis media lasted only a few days, and the labyrinthitis gradually subsided. A week after the onset echoic diplacusis appeared, sounds being heard a second later in the affected ear. Three weeks after onset the hearing was practically normal; the echoic diplacusis was gone, but the disharmonic diplacusis was still noticeable for the high tones, which were higher pitched, harsh and actually painful in the affected ear. (fig. 5).

Case 9.—Mr. D. J., aged 16, complained of earache for four days, discharge for three days and vertigo, nausea and ataxia for two days. There was spontaneous

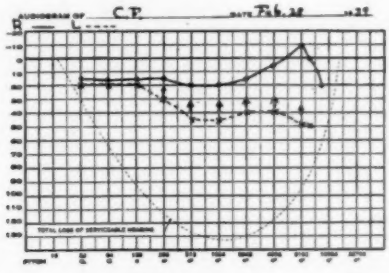

Fig. 5 (case 8).—Acute suppurative otitis media (left) with acute serous labyrinthitis and diplacusis, the 256, 512, 1024, 2048 and 4096 frequency forks being heard at a higher pitch in the left ear.

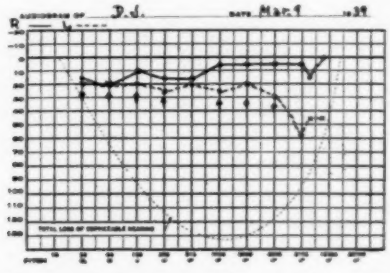

Fig. 6 (case 9).—Acute suppurative otitis media (left) with acute serous labyrinthitis and diplacusis, all forks being heard at a higher pitch in the left ear.

second degree nystagmus toward the affected ear, indicating acute serous labyrinthitis. Diplacusis was present for the 128 to 2048 vibration forks, which were heard at a higher pitch in that ear. The discharge lasted ten days; the diplacusis lasted a little longer but was gone a month later (fig. 6).

Case 10.—Miss J. M. G., aged 57, complained of defective hearing following acute suppurative otitis media eight months before. The ear had discharged for only a few days, but dizziness and ataxia with profound deafness lasted three weeks. Since then the hearing had gradually improved. Music sounded disagreeable and out of tune, but this condition also was improving. Hearing tests revealed definite diplacusis for the low tones, which were heard at a higher pitch in the

affected ear. The patient was diagnosed as having had acute serous labyrinthitis with acute otitis media.

CASE 11.—Mrs. A. B. H., aged 31, complained of a chronic discharge from the ear for six years, with attacks of vertigo, ataxia and nausea for six weeks. Two weeks before when especially dizzy she noticed that the radio sounded harsh in the affected ear. She was found to have cholesteatoma. Hearing tests showed diplacusis for all the forks, which were heard at a higher pitch in the affected ear. A radical mastoidectomy resulted in a dry ear, no more attacks of dizziness and disappearance of the diplacusis. The diagnosis was mild serous labyrinthitis complicating cholesteatoma.

CASE 12.—Mrs. M. W., aged 43, presented a chronic discharge from the ear, with a central perforation. The audiogram showed a pronounced loss for the middle tones in this ear, and the tuning fork test for these tones showed diplacusis so that they were heard at a slightly higher pitch in the discharging ear (fig. 7).

In all 5 cases of suppurative otitis media with diplacusis there was definite evidence of labyrinthine involvement. Diplacusis is an unusual finding in otitis media. Its presence indicates serous labyrinthitis.

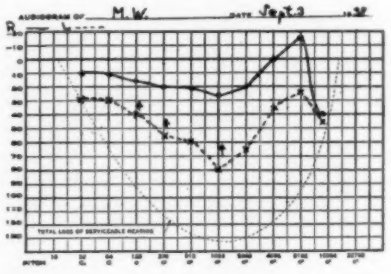

Fig. 7 (case 12).—Chronic suppurative otitis media (left) with labyrinthine deafness (evidenced by the pronounced depression of hearing) and diplacusis for the middle tones, the 128, 256 and 1024 frequency forks being heard at a higher pitch in the left ear.

PRIMARY PERCEPTION DEAFNESS WITH DIPLACUSIS (THIRTY-THREE CASES)

The great majority of the patients with diplacusis showed primary nerve deafness with nothing in the hearing tests or in the clinical examination to indicate an impairment of sound conduction. The 33 patients with this condition can be further classified etiologically as follows: 16 patients the cause of whose condition was undetermined, most of whom were seen but once; 2 patients whose deafness and diplacusis cleared up spontaneously; 2 patients in whom the onset was apoplectiform and suggested a hemorrhage into the labyrinth; 2 patients whose condition followed an acoustic trauma; 6 patients whose condition cleared up after removal of a focus of infection, and 5 patients whose

condition cleared up after removal of a specific food to which they were allergic.

Primary Nerve Deafness and Diplacusis of Undetermined Cause (Sixteen Cases)

The nerve deafness was confined to one ear or was much greater in one ear in most of the patients the cause of whose condition was undetermined. Moreover the loss of hearing usually involved the low as well as the high tones and was sometimes more pronounced for the former, so that these patients characteristically presented unilateral low tone nerve deafness with diplacusis. This unusual combination was similar to that observed in the 11 patients whose condition cleared up after removal of a focus of infection or a food allergy, so that had these 16 patients remained under observation the cause might have been determined for some of them. The following case is an example:

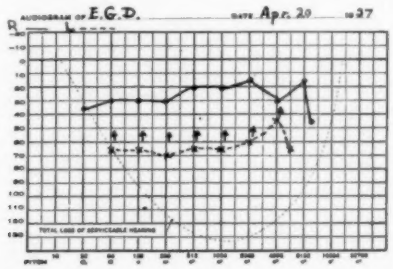

Fig. 8 (case 13).—Ménière's disease with inner ear deafness involving the low tones as well as the high tones and with diplacusis, all forks being heard at a higher pitch in the affected (left) ear.

Case 13.—Mr. E. G. D., aged 60, complained of progressive loss of hearing in the left ear for three years, with tinnitus and attacks of vertigo and ataxia for two years. Two years previously he had consulted Dr. Furstenberg, who diagnosed the condition as Ménière's disease and prescribed a salt-free diet with ammonium chloride. On this regime the dizzy attacks were less severe and less frequent but had not been completely relieved. The hearing, however, had continued to grow worse. Hearing tests revealed unilateral nerve deafness, with greater loss for the low than for the high tones and with diplacusis, all forks being heard at a higher pitch in the affected ear. Further study was advised, but the patient did not return (fig. 8).

Primary Nerve Deafness and Diplacusis with Spontaneous Recovery (Two Cases)

Case 14.—Mr. W. B. S., aged 56, complained of vertigo for six days. He was not aware of a defect in hearing, but tests showed definite lowtone nerve deafness in the left ear, with diplacusis. Three weeks later all symptoms had

cleared up. The patient during this time was under treatment for an infection of the bladder, which may have been the etiologic factor (fig. 9).

CASE 15.—Mrs. E. A. D., aged 59, a musician, complained of roaring, deafness and diplacusis in the right ear for two months. For ten months she had had a continuous cold in the head. Hearing tests revealed nerve deafness in the right ear, with loss for the low as well as the high tones and with diplacusis, all forks being heard at a higher pitch in the affected ear. Pus was obtained from the ethmoid sinuses by displacement suction. Three months later all the symptoms

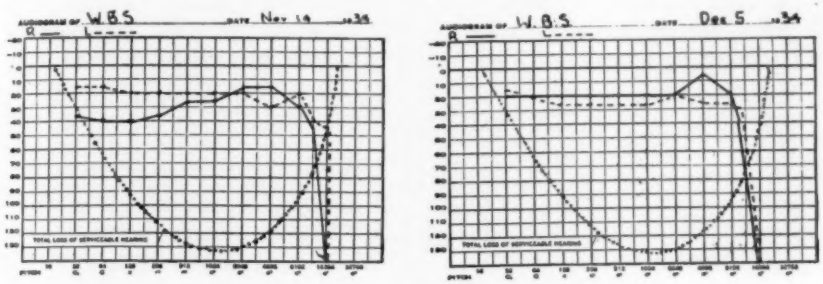

Fig. 9 (case 14).—Nerve deafness for low tones. On November 14 tests of the right ear showed that the 32 frequency fork was scarcely heard, that perception of the 64 frequency fork was slightly shortened and of the 2048 frequency fork normal and that the Rinne test was positive and perception as measured in the Schwabach test shortened. Diplacusis was present, the 32 and 64 frequency forks being heard at a higher pitch in the right ear. On December 5 tests of the right ear showed perception of the 32 and 64 frequency forks normal, and diplacusis was not present.

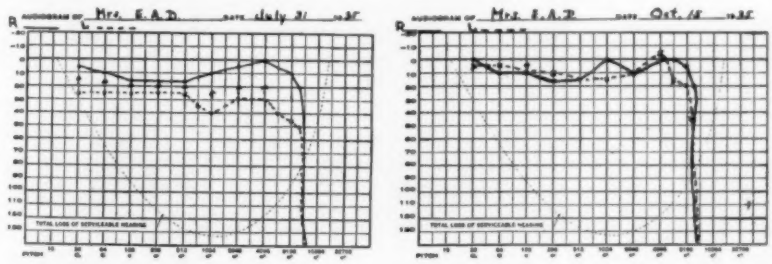

Fig. 10 (case 15).—Inner ear deafness with tinnitus and diplacusis, all forks being heard at a higher pitch in the left (affected) ear; return of hearing to normal and disappearance of diplacusis except for the 128 frequency fork; recurrence of all symptoms a week later.

had cleared up except that the 128 vibration fork was heard less than one-fourth tone lower in pitch in the affected ear. A week later the symptoms had all returned, with recurrence of diplacusis for all the forks. The patient was followed for a year, during which the hearing and diplacusis continued to fluctuate and the ethmoid sinuses continued to show evidence of infection. She was last seen two and a half years before the time of this report (fig. 10).

In retrospect the history suggests possible nasal allergy and allergy of the labyrinth. At the time, however, I was not aware of the possibility of allergy in such a disorder, and no attempt was made to discover an allergic factor.

Primary Nerve Deafness and Diplacusis with Apoplectiform Onset (Two Cases)

Case 16.—Mr. J. M., aged 19, complained of deafness in one ear for two years after concussion sustained in an accident during a game of football. The defect in hearing had not increased but had remained stationary. Hearing tests revealed nerve deafness with diplacusis, all the forks up to 1024 vibrations being

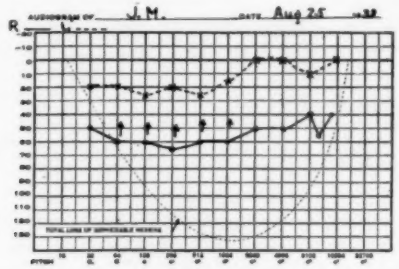

Fig. 11 (case 16).—Unilateral inner ear deafness following a blow on the head with diplacusis for the 64 to 1024 frequency forks, which were heard at a higher pitch in the affected ear (probable hemorrhage into the labyrinth).

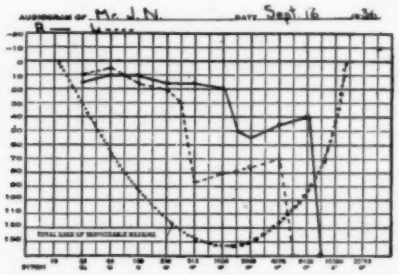

Fig. 12 (case 17).—Probable hemorrhage in the basal coil of the left cochlea. Tests of the left ear showed perception of the 32 frequency fork slightly shortened, that of the 64 frequency fork normal and the 2048 frequency fork scarcely heard. Diplacusis was present, the 64, 108, 128 and 2048 frequency forks all being heard at a slightly higher pitch in the left ear.

heard at a higher pitch and with a rough blowing quality in the affected ear. The sudden onset with concussion and the fact that the defect was stationary suggest a hemorrhage into the labyrinth with permanent distortion of the organ of Corti (fig. 11).

Case 17.—Mr. J. N., aged 70, awoke one morning deaf in the left ear with ringing, nausea, vomiting and ataxia. Previous to this his left ear had been his

better-hearing ear. During the next two months the hearing improved somewhat and then reached a level at which it has remained stationary to the time of writing. Hearing tests reveal a sharply localized defect in the left ear, with diplacusis for the low-pitched forks, which are heard at a higher pitch in that ear. The age, the sudden onset, the single attack and the subsequent partial recovery suggest a hemorrhage into the basal turn of the cochlea, destroying most of the hearing for the high tones and distorting the vibrating membrane in the apical turns so as to produce diplacusis (fig. 12).

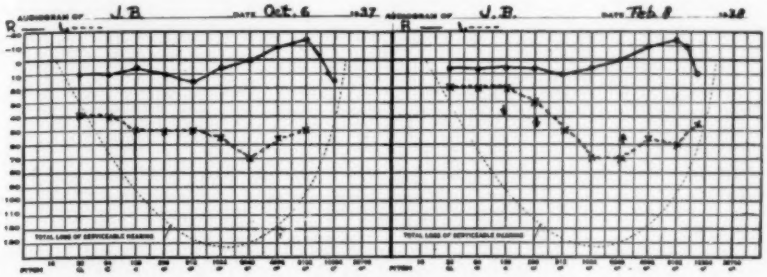

Fig. 13 (case 18).—Unilateral inner ear deafness from acoustic trauma with partial recovery of hearing and diplacusis for the 128 and 256 frequency forks, which were heard at a lower pitch in the affected ear.

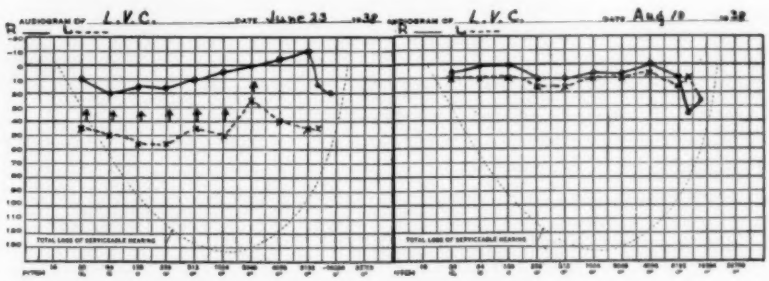

Fig. 14 (case 20).—Inner ear deafness involving low as well as high tones, with diplacusis, all forks being heard at a higher pitch in the affected ear; prompt return of normal hearing and disappearance of diplacusis, one week after the removal of a focus of infection.

Primary Nerve Deafness and Diplacusis Following Acoustic Trauma (Two Cases)

Case 18.—Mr. J. B., aged 33, complained of deafness and roaring in the left ear for ten days, which began while he was working with a new and extremely noisy machine. Hearing tests showed profound nerve deafness in that ear. Three months later the hearing for low tones had improved, but these were now heard at a lower pitch in the affected ear. The onset, the partial recovery and the diplacusis, all indicate a mechanically produced lesion of the vibrating elements in the cochlea (fig. 13).

CASE 19.—Mr. W. A. T. complained of a slight defect in hearing after a sudden loud static sound from a telephone receiver. Hearing tests gave normal results except for a slight impairment for the fork of 32 vibrations, with diplacusis, this fork being heard as a rattle rather than as a tone in the affected ear. The onset and the diplacusis suggest a mechanically produced lesion of the vibrating membrane in the apical turn of the cochlea.

NERVE DEAFNESS AND DIPLACUSIS WITH IMPROVEMENT OR RECOVERY AFTER REMOVAL OF A FOCUS OF INFECTION (SIX CASES)

CASE 20.—Mr. L. V. C., aged 33, complained of deafness and tinnitus in the left ear of three weeks' duration. Hearing tests revealed low tone nerve deafness with diplacusis, all forks being heard at a higher pitch in the affected ear. Removal of infected tonsils resulted in the disappearance of the tinnitus within four days and the return of normal hearing within a week. Previous to the tonsillectomy the deafness and tinnitus had shown no tendency to improve (fig. 14).

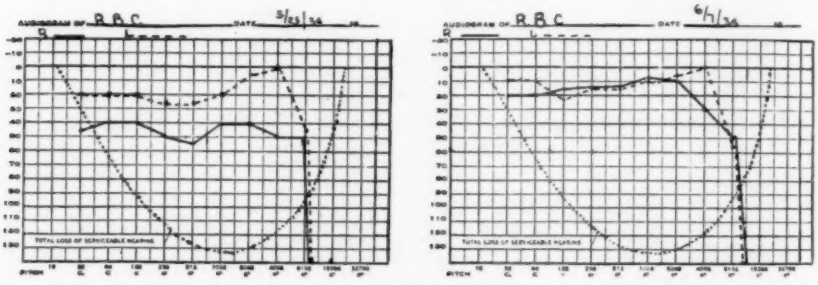

Fig. 15 (case 21).—Ménière's disease with unilateral inner ear deafness involving the low as well as the high tones, with vertigo, and diplacusis, all forks being heard at a higher pitch in the affected ear; prompt return of normal hearing one week after the removal of a focus of infection. Before tonsillectomy nerve deafness for low tones was present in the right ear. A whisper was heard at close range in the right ear and at 2 meters in the left; the 32 frequency fork was scarcely heard in the right ear, with normal perception in the left; perception of the 64 and 2048 frequency forks was slightly shortened in the right ear and normal in the left; the Rinne test was positive in both ears, and the reaction to the Weber test was lateralized to the left. Diplacusis was present, the 64, 108, 128 and 216 frequency forks being heard at a higher pitch in the right ear. One week after tonsillectomy a whisper was heard at 3.5 meters in the right ear and at 4 meters in the left; perception of the 32, 64 and 2048 frequency forks was normal in both ears; the Rinne test was positive in both ears, and the reaction to the Weber test was lateralized to the left. Diplacusis was still present, but six months later it had disappeared.

CASE 21.—Mr. R. B. C., aged 56, complained of tinnitus and deafness in the right ear, with attacks of vertigo, nausea, vomiting and ataxia for three months. Hearing tests revealed low tone nerve deafness with diplacusis, all forks being heard at a higher pitch in the affected ear. Removal of infected tonsils resulted in the complete disappearance of the tinnitus and the return of the hearing to normal within one week (fig. 15).

Case 22.—Mr. W. R. W., aged 42, complained of attacks of vertigo for five years and of tinnitus and deafness in the left ear for two weeks. An orchestra sounded distinctly out of tune. Hearing tests revealed low tone nerve deafness with diplacusis, all forks being heard at a higher pitch in the affected ear. Removal of infected tonsils resulted in normal hearing six weeks later, and there have been no more attacks of vertigo at the time of writing (fig. 16).

Case 23.—Mr. C. F., aged 42, complained of roaring and deafness in the left ear for one year. Hearing tests revealed a bilateral nerve defect, more marked in the left ear, with diplacusis, the forks being heard as a distinctly different sound in the two ears, though the patient was unmusical and could not distinguish pitch. Removal of infected tonsils resulted in an improvement in hearing almost to normal four weeks later, with the disappearance of the roaring which had been present for a year. A year later the diplacusis and impairment of hearing recurred during a sinal infection. The patient was then lost track of (fig. 17).

Case 24.—Miss U., aged 40, complained of high-pitched tinnitus in the left ear for seven weeks. The hearing was normal, and the cause of the tinnitus was

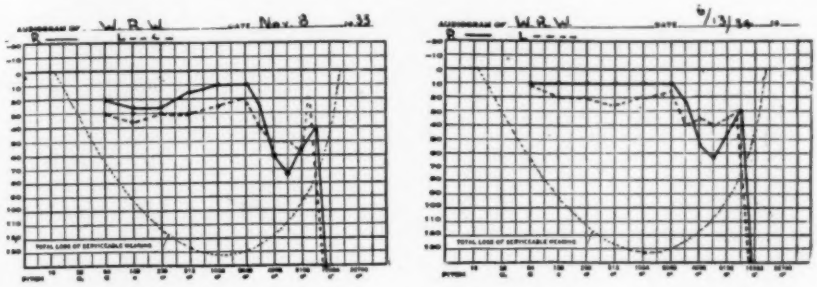

Fig. 16 (case 22).—Ménière's disease with inner ear deafness involving low as well as high tones and diplacusis, all forks being heard at a higher pitch in the affected ear; complete recovery six weeks after the removal of a focus of infection. Nerve deafness for low tones was present on the left; the 32 frequency fork was not heard, and perception of the 64 frequency fork was moderately, and that of the 2048 frequency fork slightly, shortened. The reaction to the Weber test was lateralized to the right; the Rinne test was positive in the left ear, and the reaction to the Schwabach test was normal. Diplacusis was present for all tones, the sounds appearing of poor quality and lower in pitch in the left ear. Six weeks after tonsillectomy perception of the 32 and 64 frequency forks was normal and that of the 2048 frequency fork slightly shortened on the left. Diplacusis was not present.

not found. Three months later she returned because of an increase in the tinnitus, with a distinct jarring discomfort in the affected ear when it was exposed to loud sounds. Hearing tests now disclosed low tone nerve deafness in that ear, with diplacusis for the low-pitched forks. Removal of two dead teeth, one of which proved to be abscessed in spite of a normal roentgenogram, resulted in marked improvement in the tinnitus within a week and complete recovery soon afterward. Three months later the symptoms returned, with three attacks of vertigo, but they cleared up spontaneously within a few weeks and have not recurred at the time of writing (fig. 18).

Case 25.—Mrs. M. A. W., aged 70, complained of nasal symptoms, which proved to be due to chronic maxillary sinusitis. For six or seven years she had had defective hearing and roaring in the right ear. Three years before I saw her she had had a series of attacks of severe vertigo, nausea, vomiting and ataxia. She had visited a university clinic, where an audiogram was obtained; the tuning fork tests showed unilateral nerve deafness, and she was placed on a salt-free diet, with relief from the attacks of dizziness. Hearing tests when I saw her showed low tone nerve deafness with diplacusis, the low forks being heard at a higher pitch in the affected ear. The interesting thing is that clearing up the chronic maxillary sinusitis by a series of irrigations resulted in the restoration of hearing nearly to normal (fig. 19).

Five of the 6 cases of low tone nerve deafness and diplacusis with recovery after removal of a focus of infection have been reported previously. As was pointed out at that time, the age of the patients, the response to removal of a focus of infection and the frequent tendency to recurrences, all suggest a similarity to iritis. It was suggested that

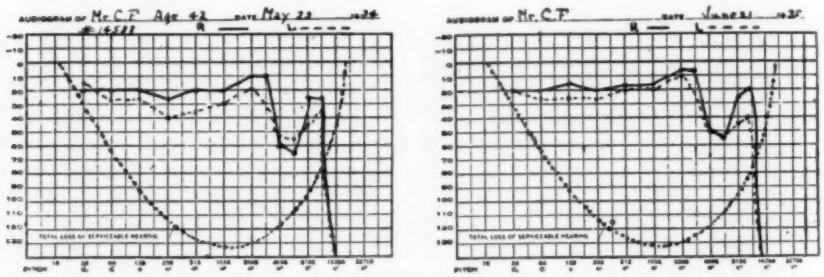

Fig. 17 (case 23).—Inner ear deafness involving low as well as high tones and diplacusis, all forks being heard at a higher pitch in the affected ear; return of normal hearing four weeks after the removal of a focus of infection. Before tonsillectomy nerve deafness for low tones was present in the left ear, with perception of the 32 frequency fork much shortened and that of the 64 and 2048 frequency forks slightly shortened; the Rinne test was positive in the left ear, and perception as measured in the Schwabach test was shortened. Diplacusis was present for the 64, 108 and 128 frequency forks, but the patient was not musical and could not say whether the sound was higher or lower in the affected ear. Four weeks after tonsillectomy perception of the 32, 64 and 2048 frequency forks was normal in the left ear. Diplacusis was present for the 64, 108 and 128 frequency forks.

the pathologic change in these cases is an inflammatory process in the labyrinth caused by focal infection elsewhere in the body. This inflammatory change results in edema or thickening of the vibrating membrane, thus causing the diplacusis.

Primary Nerve Deafness and Diplacusis with Improvement or Recovery After Removal of an Allergic Factor (Five Cases)

Case 26.—Mr. D. A. complained of tinnitus and increasing deafness in the left ear for many years, with attacks of vertigo, nausea, vomiting and ataxia

for four years, gradually increasing in frequency. A salt-free diet with ammonium chloride had failed to relieve the dizziness. Hearing tests disclosed low tone nerve deafness with diplacusis, all forks being heard at a higher pitch and with a buzzing quality in the affected ear. The nasal smear revealed a definite increase in eosinophils. An elimination diet excluding the common causes for food allergy, such as wheat, milk, eggs, and chocolate, resulted in complete relief from the dizziness. The addition of wheat was followed by a recurrence of the vertigo.

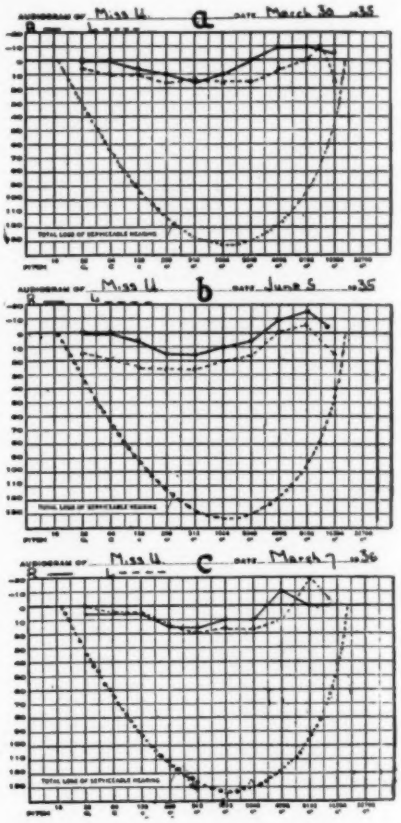

Fig. 18 (case 24).—Inner ear deafness involving low more than high tones and diplacusis for the low tones, which were heard at a higher pitch in the affected ear; recovery of normal hearing one week after the removal of a focus of infection. At the first examination the hearing was normal, with perception of the 32 frequency fork very slightly shortened in the right and slightly shortened in the left ear and that of the 64 and 2048 frequency forks normal in both ears (*a*). Later, nerve deafness for low tones and diplacusis were present in the left ear; perception of the 32 frequency fork was very slightly shortened in the right ear, and the fork was scarcely heard in the left; perception of the 64 frequency fork was normal in the right ear and moderately shortened in the left, and that of the 2048 frequency fork was normal in both ears (*b*). After operation, normal hearing returned, and the diplacusis largely disappeared; perception of the 32, 64 and 2048 frequency forks was normal in both ears (*c*).

The addition of other foods and the exclusion of wheat has resulted in permanent relief from the dizziness. The hearing has improved slightly, but the diplacusis persists (fig. 20).

Case 27.—Col. G. V., aged 47, complained of tinnitus in the right ear for six weeks and an attack of dizziness and nausea eight days previously. Hearing tests revealed slight low tone nerve deafness in the affected ear, with diplacusis, the low-pitched forks being heard at a higher pitch and with a peculiar vibrating quality in that ear. The nasal smear showed an increased proportion of eosinophils,

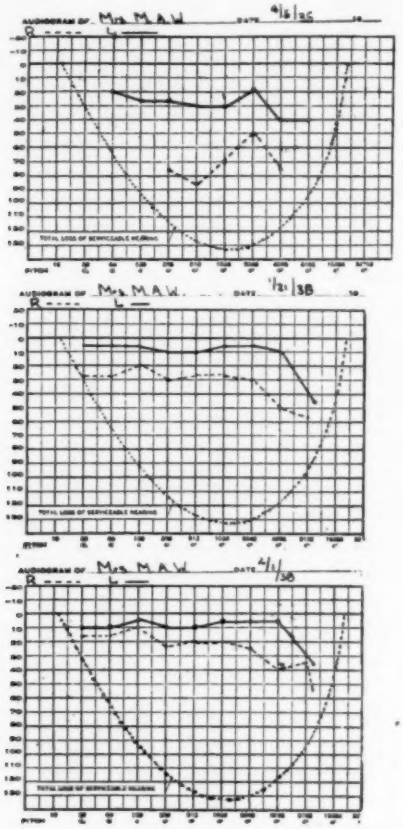

Fig. 19 (case 25).—Ménière's disease with a profound defect in hearing and diplacusis, the low-pitched forks being heard at a higher pitch in the affected ear; marked improvement in hearing, nearly to normal, after removal of a focus of infection.

and there were symptoms of chronic rhinitis. Because wheat is the most frequent cause for food allergy in the upper respiratory tract it was removed from the diet. The chronic rhinitis, which had lasted four months, and the tinnitus and diplacusis, present for six weeks, cleared up completely within two weeks.

Case 28.—Miss E. L., aged 58, complained of tinnitus and gradual impairment in hearing in the left ear, with attacks of vertigo, nausea, vomiting and

ataxia for one year. Hearing tests revealed low tone nerve deafness in the left ear, with diplacusis, all forks being heard at a higher pitch in that ear. A diet with a low salt content with ammonium chloride failed to relieve the dizziness. An elimination diet omitting wheat, eggs, chocolate, nuts, onions, garlic and condiments resulted in complete relief. The addition of eggs was followed by a recurrence. At the time of writing the patient is omitting eggs and the other foods enumerated and prefers to follow this diet rather than to experiment with adding foods and running the risk of more spells of dizziness.

Case 29.—Mrs. F. T. M., aged 23, complained of tinnitus and deafness in the left ear, vertigo, nausea, vomiting and ataxia, all for three days. For two years she has experienced periods of impaired hearing in the left ear, not related to colds in the head. Hearing tests revealed low tone nerve deafness in the left ear, with diplacusis confined to the 2048 vibration fork, which was heard three or four tones higher in pitch in that ear. There was spontaneous nystagmus toward the affected ear. Because she suspected that coffee and orange juice disagreed with her and because the attack of dizziness followed the ingestion of an unusual amount of coffee, the patient was advised to omit coffee and orange

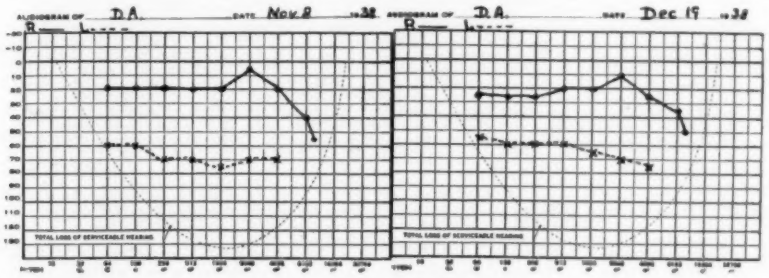

Fig. 20 (case 26).—Ménière's disease with inner ear deafness involving low as well as high tones and diplacusis, all forks being heard at a higher pitch in the affected ear. No improvement on Furstenberg's regimen; complete freedom from vertigo on removing wheat from the diet, with recurrence on adding wheat; slight improvement in hearing.

juice from her diet. The symptoms improved greatly but recurred when she took a cup of coffee a week later. At the time of writing she has been relatively free from aural symptoms for five months.

Case 30.—Mrs. P. W. P., aged 50, complained of roaring and deafness in the left ear for a year. Removal of an infected remnant of a tonsil and of several infected teeth resulted in marked improvement in the hearing, but soon the symptoms returned. She complained that her husband's voice sounded harsh and disagreeable in the affected ear, and hearing tests revealed low tone nerve deafness with diplacusis, the low-pitched forks being heard at a higher pitch in that ear. The hearing fluctuated widely for the next two months. She then experienced a sudden attack of vertigo, nausea, vomiting and ataxia. Removal of infected lateral pharyngeal bands was followed by improvement in the hearing and the roaring. For a year she remained free from aural symptoms, and then the tinnitus, low tone nerve deafness and diplacusis, all recurred. No focus of infection could be found, and the deafness and tinnitus persisted for six months. Finally an elimination diet was advised, the foods commonly causing allergy being excluded.

Within a few days the hearing, which had been poor for six months, improved markedly. The addition of milk was followed within a few hours by a red, sore rash in the mouth, with puffiness of the lower lip. The patient then mentioned that ingestion of milk and cream had often resulted in a sore mouth. Milk, cream, butter and cheese were therefore excluded and all other foods added to her diet, and on this regime the hearing returned to and remained normal. To prove definitely that allergy to milk was the cause of the aural trouble it was suggested to the patient that she add milk to her diet for a week. This was done and resulted in profound deafness, much worse than she had ever had before, with a discharge of thin serum from the middle ear, which began painlessly and lasted ten days. Milk, cream, butter and cheese were immediately removed permanently from her diet. The otitis media cleared up, and the hearing slowly returned to normal, at which it has remained (fig. 21).

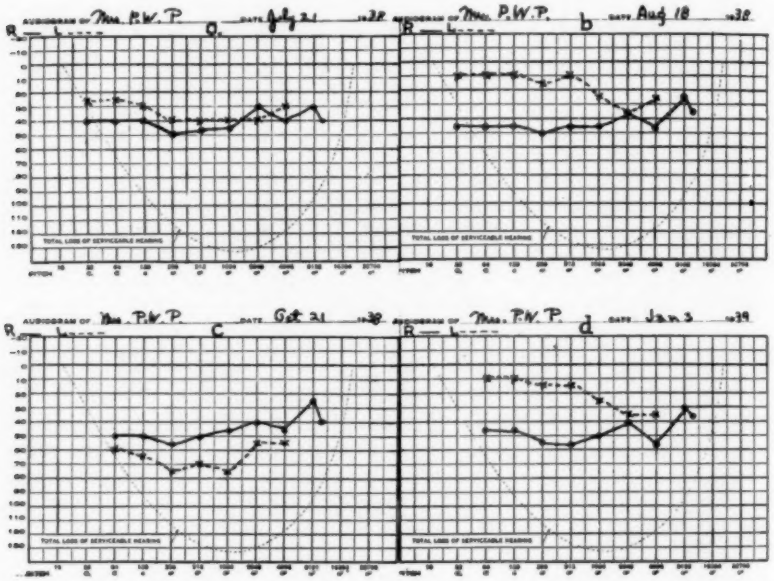

Fig. 21 (case 30).—Inner ear deafness (left) involving low as well as high tones and diplacusis, all forks being heard at a higher pitch in the left ear, with a conduction defect (right) due to a total perforation of the drum membrane (*a*); return of hearing to normal on removing milk from the diet (*b*); profound inner ear deafness within a few days of adding milk to the diet (*c*); return of normal hearing after excluding milk from the diet again.

COMMENT

Analysis of the 45 patients with diplacusis shows that in every case, except that of the patient with normal hearing, there is evidence of a lesion in the inner ear. In the few cases of otitis media with diplacusis there was always associated acute serous labyrinthitis. Clinical experience, therefore, confirms the theoretic statement that diplacusis can be caused only by a lesion of the organ of Corti.

Attacks of vertigo occurred in many of the patients with inner ear deafness and diplacusis so that the cases may be regarded as instances of Ménière's disease (cases 13, 14, 21, 22, 24, 25, 27, 28, 29 and 30). Since including the test for diplacusis in the routine hearing tests I have found the symptom in the great majority of patients with Ménière's syndrome. This means that the lesion of Ménière's disease is in the membranous labyrinth. In some cases it is apparently the result of focal infection (cases 21, 22, 24 and 25). As Crowe [5] pointed out, one must be guarded in assessing the value of any therapeutic measure for Ménière's disease, because of the spontaneous remissions that occur so often. However, in case 21, in which the tinnitus and deafness had persisted for three months only to clear up completely within a week after tonsillectomy, it is difficult not to conclude a causal relation. In case 22 mild dizzy spells of five years' duration completely ceased after tonsillectomy, a result that seems more than a coincidence. Wright [6] has pointed out also that Ménière's disease may be caused by focal infection, and he noted the similarity between the clinical course of iritis and of Ménière's disease, a similarity which I pointed out several years earlier.[7]

It seems unlikely that in all cases Ménière's disease is due to focal infection. Allergy causes it in at least some cases (cases 26, 27, 28, 29 and 30). Here again one must be guarded in interpreting the results of therapy. Conclusive proof of an allergic cause is obtained by relief from symptoms when the substance is avoided and a recurrence of symptoms on exposure to the substance followed again by relief on its removal. Such proof was obtained in cases 26 and 30.

The idea that allergy may be responsible for Ménière's disease in some cases is not new. Duke, Kobrak, Proetz, Yandell, Vaughn and Hawke, Vogel, Urbach and Wilder, Malone, Rowe and Richet, were all cited by Dean, Agar and Linton [8] as reporting cases of Ménière's disease due to allergy. The latter authors report a series of additional cases. Jones [9] described an instance of Ménière's disease due to allergy to milk. Campbell [10] and Dohlman [11] observed cases of Ménière's diseases due to allergy to specific foods.

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5. Crowe, S. J.: Ménière's Disease, Medicine **17**:1 (Feb.) 1938.

6. Wright, A. J.: Aural Vertigo: Clinical Study, J. Laryng. & Otol. **53**:97 (Feb.) 1938.

7. Shambaugh, G. E., Jr.: The Significance of Diplacusis in Ménière's Syndrome, in Medical Papers Dedicated to Henry Asbury Christian, Baltimore, Waverly Press, Inc., 1936.

8. Dean, L. W.; Agar, J. S., and Linton, L. D.: Allergic Diseases of the Ear, Laryngoscope **47**:707 (Oct.) 1937.

9. Jones, M. F.: Manifestations of Allergy in the Ear, Ann. Otol., Rhin. & Laryng. **47**:910 (Dec.) 1938.

10. Campbell, P.: Personal communication to the author.

11. Dohlman, G.: Personal communication to the author.

The first histologic study of the ear in the presence of Ménière's disease was made recently by Hallpike and Cairns.[12] They observed extreme and uniform dilatation of the endolymphatic system. They expressed themselves as unable to explain this increase of endolymph but suggested that sudden changes in pressure of the endolymph cause anoxemia of the labyrinth. They suggested that stretching of the basilar membrane might lead to a change in its vibration and diplacusis. Since these observations were made on patients who died in the interim between attacks, it is conceivable that the picture observed by the authors is the residuum after acute attacks of allergic edema of the membranous labyrinth. The peculiar fluctuating nature of the deafness in Ménière's disease pointed out by Crowe [5] is consistent with an allergic disorder or with an inflammatory lesion resulting from a focus of infection. The beneficial results obtained by Furstenberg and his co-workers [13] by a restricted sodium-low diet and rest in bed for Ménière's disease are not entirely inconsistent with an allergic disorder, while the finding of subcutaneous edema in patients with Ménière's disease by Mygind and Dederding [14] is suggestive of allergy.

A word should be said concerning the inner ear deafness for low tones which I have observed in patients with Ménière's disease and diplacusis. The loss of hearing for low tones may be as great as or greater than that for high tones. This has led Mygind and Dederding to the statement that, although the patients have shortened perception by bone conduction, they have middle ear deafness and that, therefore, shortened perception as measured in the Schwabach test does not indicate inner ear deafness! My reasons for believing that the defect in perception of low tones in such cases is due purely to a lesion in the inner ear are as follows:

1. Perception as measured in the Schwabach test is always shortened.

2. When reaction to the Weber test is lateralized, the sound is always heard in the better hearing ear.

3. The Rinne test remains positive even with a considerable defect in the affected ear.

4. The drum membrane and eustachian tube are normal, and inflation does not result in demonstrable improvement in hearing.

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12. Hallpike, G. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625 (Oct.) 1938.

13. Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ménière's Symptom Complex: Medical Treatment, Ann. Otol., Rhin. & Laryng. **43**:1035 (Dec.) 1934.

14. Mygind, S. H., and Dederding, D.: Ménière's Disease as an Indication of Disturbances in the Water Metabolism, Capillary Function, and Body Condition, Ann. Otol., Rhin. & Laryng. **47**:55 (March) 1938.

5. Marked fluctuations in hearing are not accompanied by visible or demonstrable changes in the middle ear or eustachian tube.

Crowe [5] did not mention the loss of hearing for low tones, but his audiograms clearly show it. He and Wright agreed in stating that the defect in hearing in Ménière's disease is a type of inner ear deafness.

Disease of the membranous labyrinth may be confined to the vestibular portion; it may involve both the vestibular apparatus and the cochlea, or it may affect the cochlea alone. In the first instance there will be vertigo without deafness; in the second, vertigo with deafness, and in the third, deafness without vertigo. The disease, however, remains essentially the same, namely, a chronic or recurrent involvement of the membranous labyrinth. This disease is characterized by the following peculiarities which constitute a symptom complex or syndrome:

1. Attacks of vertigo.

2. Inner ear deafness with loss of hearing for the low tones as often as for the high tones and with a tendency to marked fluctuations.

3. Diplacusis with a tendency to fluctuate.

4. Tinnitus which tends to fluctuate.

The etiology of this chronic disease of the membranous labyrinth is not yet entirely clear. Focal infection seems to be a factor in some cases. Allergy is definitely the cause in some cases. The term "Ménière's disease" is not satisfactory for this chronic disease of the labyrinth, since it refers only to attacks with vertigo. Some term should be used to include the attacks without vertigo but with the other symptoms of the syndrome. "Chronic nonsuppurative labyrinthitis" might be a suitable term except when a specific allergy is proved, in which event "allergic labyrinthitis" is the proper term.

CONCLUSIONS

1. Diplacusis is due to a lesion of the organ of Corti.

2. Diplacusis is a more frequent symptom than is generally appreciated, but to be detected it must be specifically tested for.

3. The test for diplacusis is simple, quick and reliable. It is best done with tuning forks.

4. Diplacusis occurs in the following clinical conditions:

   (*a*) Otosclerosis with labyrinthine involvement (cases 3 and 4).

   (*b*) Acute serous labyrinthitis secondary to acute or chronic suppurative otitis media (cases 8, 9, 10, 11 and 12).

   (*c*) Operative trauma to the membranous labyrinth (cases 5, 6 and 7).

   (*d*) Hemorrhage into the labyrinth (probably cases 16 and 17).

(*e*) Acoustic trauma to the labyrinth (cases 18 and 19).

(*f*) Labyrinthitis resulting from focal infection (cases 20, 21, 22, 23, 24 and 25).

(*g*) Labyrinthitis due to allergy (cases 26, 27, 28, 29 and 30).

5. Diplacusis is found in most cases of Ménière's disease with defective hearing.

6. Ménière's disease is due to nonsuppurative labyrinthitis, sometimes allergic, sometimes the result of focal infection. There may be vertigo without deafness, vertigo and deafness or deafness without vertigo, according to whether the entire membranous labyrinth or only a portion is involved.

7. Perception deafness due to a lesion of the membranous labyrinth is characterized by the following conditions:

(*a*) The hearing for low tones is impaired as frequently as that for high tones.

(*b*) Diplacusis is present.

(*c*) Vertigo is frequent.

(*d*) In many cases, the pathologic lesion is reversible, at least in the early stages, so that the hearing is subject to marked fluctuations, and it may return to normal if the etiologic factor is removed.

ABSTRACT OF DISCUSSION

Dr. Arthur W. Proetz, St. Louis: Dr. Shambaugh has observed the cases over a long period. Diplacusis has been understood and described for several generations, but it has never occurred to anybody to deduce from observations that the fault lay in the cochlea and not somewhere along the line, as might have been deduced provided that the Helmholtz resonator theory is accepted.

Knowingly or not, Dr. Shambaugh has contributed a bit of evidence in favor of the resonator theory. In each case in his list, with one exception, the pitch was raised, no matter for what part of the scale the perception was affected.

The physical laws of vibrating bodies are well known. The fundamental resonance depends on length and mass and volume, and if the lesion can be conceived as a swelling of the organ of Corti naturally all these factors are increased and the tone would be expected to rise in pitch.

It seems to me that if there were some infection of the central apparatus the deviation might occur in any direction. It is hard to think that a lesion would extend only in such a direction, centrally, as to produce a perception of higher pitch and never a lower pitch. With a lesion in the cochlea, this is easily understood.

As Dr. Shambaugh says, diplacusis is not rare. I experienced it a few years ago myself and so became interested in it. After an acute attack of otitis media, so acute that the drum ruptured before I could get any help, the diplacusis appeared, beginning at 1024 vibrations and getting worse as the pitch rose, to 16000 vibrations, at which there was a difference of four and a half tones between the true

pitch and that heard in the infected ear; that is, F sharp was heard instead of C. Fortunately, two audiometers, one with a continuous range of pitch, were at hand, so that the phenomenon could be studied. It lasted twelve days. But nowadays I do not use a tuning fork for testing diplacusis, and I find the best means at my disposal is a two way double ear phone, now to be had from at least one audiometer manufacturer, which was originally intended as a test for malingering. The switch is so arranged that the tone can be given first to one ear, then gradually to both and finally to the other, without variation in pitch or in intensity. If this double earphone is given a patient and a certain tone is sent at a certain volume through the audiometer, the patient will report hearing either one tone or two tones, and it is especially valuable for persons who have no musical sense. There is a particular facility that one can develop in eliciting the symptom from such persons. A musician will say at once, "I hear a triad," or " I hear a dissonance," or "I hear a consonance" or something else besides the one tone that the examiner is making. The person who is not musical simply says, "It is a rasping noise."

It is, after all, a dissonance produced by a slight rise in the pitch perceived in one ear.

Dr. Shambaugh has been modest in confining his conclusion to the localization of the lesion in the cochlea. With further study he may be able to localize it still further, because obviously in some cases the high pitches are involved and in other cases the low pitches, which may give an even better understanding of the suggestion. As a matter of fact, it is in cases of inflammation, usually, that the high pitches are affected, as might be expected from the positions of the various turns of the cochlea.

Dr. George N. Coates, Philadelphia: I am not in the fortunate position of Dr. Proetz, who can discuss this interesting paper from firsthand knowledge, having experienced the condition himself. Most otologists are compelled to depend on the patient's own statements that there is a disharmony in the tones perceived in the two ears in diplacusis binauralis dysharmonica, which is the common type and, in fact, the only type with which they are familiar.

I strongly suspect that the majority of otologists do not routinely search for this symptom, but only notice it when it is specifically called to their attention by an unusually observant patient. Such patients in my experience have been musicians or at least devotees of symphonic or other high class music, in other words, those who have musically trained ears.

From Dr. Shambaugh's interesting case reports, it is evident that otologists should be more careful in their investigations in this respect and that if they are they will have another valuable aid to diagnosis and prognosis. I further suspect that if they make a routine practice of this type of testing, they will be rewarded by finding the symptom in more cases than they formerly thought likely.

Without any real study of the subject, I have always assumed that diplacusis must be due to some pathologic condition in the labyrinth, temporary or permanent, which in some way changed the vibrating mechanism in the organ of Corti. This is indicated by the deductions that the author has drawn from a rather large series of cases, in all of which, irrespective of the causation, labyrinthine involvement and perceptive deafness for either high or low tones and in many of which vestibular symptoms, appeared. Even in the cases of otosclerosis the condition at least started with, or was complicated by, cochlear otosclerosis, and the cases in which the symptom was observed after fistulization are especially interesting.

As for the diagnosis, I believe that Dr. Shambaugh is quite right in saying that careful testing with the tuning forks over the whole series of octaves to at

least 2048 double vibrations is the best if not the only way to confirm the patient's statements, and this takes time and patience. The method outlined is admirable, particularly if the person being tested is able to reproduce the different pitch heard in each ear. When this is done, the examination is conclusive.

In respect to the probability of pathologic change in the cochlea as the cause of this condition, one must wait for pathologic diagnosis at autopsy before one can be sure. In the case of trauma, whether acoustic or with hemorrhagic distortion of structure by direct violence or by the formation of cicatrices seems a ready explanation. Such occurrences should be accompanied by permanent loss of hearing in most instances, and it seems that the diplacusis also might be permanent. When the condition is attributable to allergy or to focal infection, it may well be that serous labyrinthitis or edema, similar to that seen in other parts of the body in the same cases, causes the distortion in the membranous labyrinth that causes the disharmony. Clinical results certainly seem to corroborate this thesis, since on the removal of the source of infection or of the offending allergens not only does other edema subside but the impaired hearing, the vestibular symptoms and the diplacusis improve as well.

I wish that Dr. Shambaugh in his closing discussion would amplify the remarks contained in the last paragraph of his paper relative to the differential diagnosis and prognosis in cases of nerve deafness.

Dr. George E. Shambaugh Jr., Chicago: The use of the special audiometer described by Dr. Proetz for detecting diplacusis is helpful. That type of audiometer, however, is certainly not available to most otologists. I use a 1-A audiometer with which the sound can be shifted from one ear to the other, but it is extremely difficult to get the intensity of the tone equal in both ears. With a tuning fork that is done easily. One holds the fork in front of the normal and then the affected ear, regulating it until the intensity is equal. Few persons can distinguish between pitch and intensity. One has to get the intensity exactly equal and then ask about the pitch.

Dr. Coates asked me to amplify the last paragraph, concerning the differential diagnosis of the two types of nerve deafness. In most cases nerve deafness, as illustrated by the senile type or the type which follows prolonged exposure to loud noises, consists essentially, pathologically, of degeneration of the cells of the spiral ganglion. That type of lesion is not reversible; once it has occurred, the deafness is permanent, and treatment has no effect. A lesion involving the vibrating membrane in the organ of Corti, however, particularly if it is an inflammatory or an allergic edema, is essentially reversible, and if the cause is determined and removed before permanent changes have occurred the hearing should return to normal.

122 South Michigan Avenue.

Case Reports

ADENOCARCINOMA ARISING FROM THE MUCOUS GLANDS OF THE PALATE WITH PULMONARY METASTASES

J. G. Sharnoff, M.D., and J. R. Lisa, M.D., New York

The purpose of this report is to present a case of true adenocarcinoma of the palate originating in the mucous glands with metastases to the regional lymph nodes and the lungs. The term "true" is used in order to differentiate this tumor from the several tumors which have been reported in the literature as adenocarcinoma by Ribbert,[1] Boenninghaus,[2] Coenen [3] and others. The latter neoplasms were designated by these authors as adenocarcinomas, although clinically and histologically they fall into the controversial group of relatively benign tumors more commonly referred to by others as cylindromas. These authors stated the belief that the tumors were really epithelial, as first suggested by Ribbert. The tumor that is described in this report differs clinically as well as histologically from those mentioned. Other reports in the literature refer only to squamous cell epitheliomas or mixed tumors of the palate. New and Childrey [4] in a report of 220 malignant growths of the palate seen at the Mayo Clinic from 1917 to 1930 failed to find 1 instance of adenocarcinoma.

REPORT OF A CASE

A Negress 39 years old was admitted to City Hospital, otolaryngologic service of Dr. O. C. Risch, on Feb. 2, 1939, with increasing difficulty in speech of nine months' duration, increasingly severe cough, pain in the chest and shortness of breath for several weeks. She stated that she had been treated at the Vanderbilt Clinic since June 1938. A report from that institution stated that the patient had noted a hard tender mass in the neck and soreness of the throat for about two months. It was noted that she had a nasal quality to the voice. She gave a history of having received injections in the arm and hip for more than a year. Examination revealed a destructive lesion of the roof of the mouth, which was difficult to see because of trismus. The soft and hard palates were destroyed in the midline as far forward as the second molar tooth. A lesion measuring 2 by 2.5 cm. contained a perforation 3 mm. in diameter in its center. The edge was red and

From the Pathological Laboratory, City Hospital, Welfare Island, Department of Hospitals.

1. Ribbert, H.: Geschwulstlehre für Aerzte und Studierende, ed, 2, Bonn, F. Cohen, 1914, pp. 591-601.

2. Boenninghaus, G. K. M.: Der Drüsenkrebs des harten Gaumens, Beitr. z. klin. Chir. **111**:215, 1918.

3. Coenen, H.: Ueber Gaumengeschwülste, Arch. f. klin. Chir. **75**:542, 1904.

4. New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx: Three Hundred and Fifty-Seven Cases; Malignant Tumors Exclusive of Adenocarcinoma of the Mixed Tumor Type: Two Hundred and Twenty Cases, Arch. Otolaryng. **14**:713 (Dec.) 1931.

somewhat granular. Considerable mucus was present in this area. The cervical nodes on the left were enlarged and hard, the largest in the region of the angle of the jaw. A Kline test gave a 3 plus reaction. A specimen taken for biopsy from the soft palate was reported as showing carcinoma with the comment, "Whether this is a metastatic carcinoma or a primary carcinoma arising from the mucous glands it is impossible to say from this section." Roentgen examination of the chest revealed evidence of metastases. The patient was given extensive radiotherapy to the soft palate and neck, as well as palliative therapy to the chest.

Fig. 1.—Section from the palate revealing the morphologic picture and the invasive character of the primary tumor.

Physical examination on the patient's admission to the City Hospital revealed an emaciated, chronically ill Negress with dyspnea and orthopnea. The essential physical findings were a clean, punched-out perforation in the midline of the palate and a few firm, somewhat enlarged, moderately fixed cervical lymph nodes. The examination of the chest revealed slight dulness to percussion and patches of fine crackling rales over the right anterior thoracic wall. A few scattered rales were heard at both bases posteriorly. The other physical findings were essentially negative except for the marked emaciation. A roentgenogram of the chest revealed

bizarre shadows in the lower half of the left pulmonary field suggestive of metastases. The Wassermann reaction of the blood was 4 plus. A clinical diagnosis of bronchopneumonia and carcinoma of the palate with metastases to the lymph nodes and the lungs was made. The patient died on February 6.

Necropsy.—Only the pertinent findings are abstracted. The body was that of a moderately well developed and markedly undernourished Negress. The mouth was opened with difficulty. A firm mass could be felt in the soft palate extending to

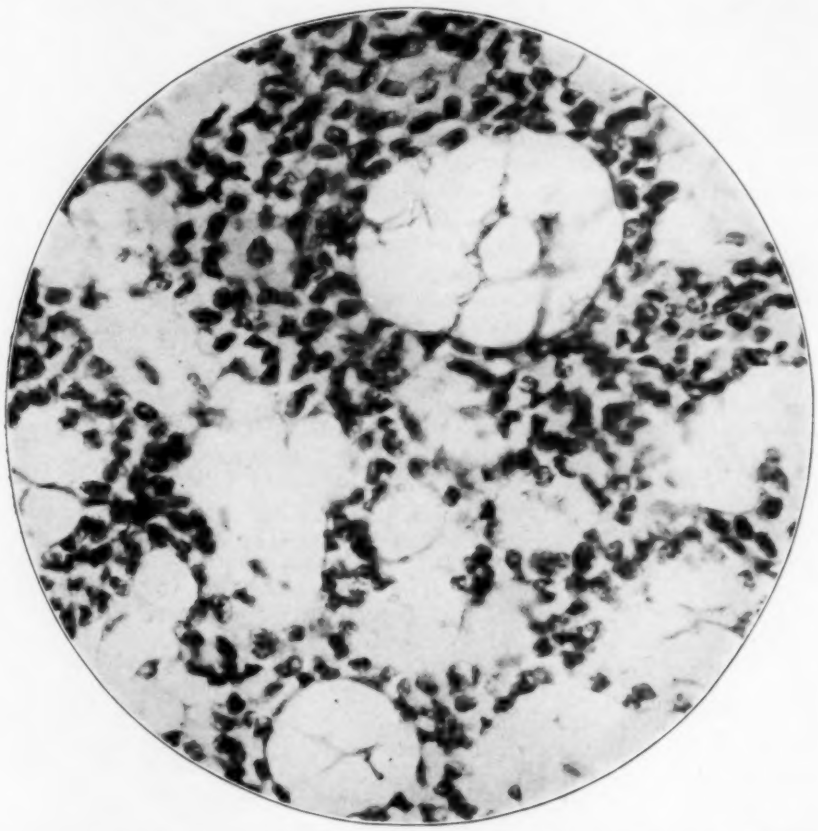

Fig. 2.—Section from the lung showing the morphologic picture of the metastases under higher magnification. The mucoid secretion is especially noticeable and prominent.

the left lateral wall of the pharynx. A small perforation with a regular smooth edge was noted to the left of the midline in the soft palate. The pleural cavities were free of fluid, the surfaces smooth, glistening, moist and free of adhesions. The lungs were moderately voluminous. Several round or irregularly oval firm elevated areas of lighter color than the surrounding slate gray lung were noted. On section these areas were circumscribed, nodular, and rubbery with a gray dry surface. They were gray-white with moderate anthracotic markings. These areas were scattered throughout all the lobes, chiefly at the periphery of the lower

lobe of the right lung. Several smaller nodules nearer the hilus were lying close to small blood vessels. In addition there were a few small recent infarctions with thrombosis of the arteries.

Histologic Picture.—Sections of the soft palate revealed a normal stratified squamous epithelium covering a dense irregular connective tissue stroma moderately infiltrated by lymphoid and plasma cells. Some areas presented a normal striated musculature and many islands of normal mucous glands. Adjacent to the latter areas, embedded in the dense connective tissue, invading and partially destroying the musculature were partly solid, partly alveolar cords of small epithelial cells. The epithelial cords were markedly irregular and compressed by the surrounding connective tissue. Many of the cords revealed successful attempts to form irregular acini. The acini contained a light blue–staining mucoid material. The small cells had small hyperchromatic nuclei and varied greatly in size and shape. Many were round or oval. Atypical mitoses were not noted.

Sections from the various lobes of the lungs were similar in appearance. Irregular nodules composed of cords of epithelial cells similar to those described were lying in a connective tissue stroma about the larger blood vessels and bronchioles. These cords irregularly invaded the adjacent edematous pulmonary parenchyma. There was a far greater tendency to form acini, with greater quantities of mucoid material in the various nodules.

The pathologic diagnosis was adenocarcinoma arising from the mucous glands of the palate with pulmonary metastases.

In this case are presented clinical as well as histologic criteria of malignant tumor of the mucous glands of the palate, in contrast to the only other cases reported in the literature as adenocarcinoma of the palate, by Ribbert, Boenninghaus, Coenen and others. When first seen, the patient already had a perforated ulcerating lesion of the palate with roentgenologic evidence of pulmonary metasases and clinical evidence of metastases to the cervical lymph nodes. These findings were substantiated at autopsy. The rapid course of the disease also is of significance. The growths reported as adenocarcinoma by the authors cited all began as large masses in the palate covered by normal mucous membrane, were usually of years' duration and did not recur after excision, and the reports do not indicate whether metastases ever occurred. Histologically these growths resemble those originally described by Billroth as cylindroma or by the French observers as "adenome."

SUMMARY

A case of adenocarcinoma of the palate arising from mucous glands with metastases to the cervical lymph nodes and the lungs is presented. Search of the literature failed to discover a report of a case of a metastasizing tumor of this character.

RELIEF OF NEURITIS OF EIGHTH CRANIAL NERVE WITH VITAMIN B_1

K. C. Brandenburg, M.D., Long Beach, Calif.

Among recent therapeutic agents which are showing startling successful results in conditions which have hitherto responded to treatment slowly or not at all, synthetic vitamin B_1 deserves closer attention than it is receiving in the field of ophthalmology and otorhinolaryngology. My purpose in presenting this case report is to direct attention to the possibilities of this new form of therapy, in the hope that its application may be extended to other conditions in which it is likely to prove of benefit.

F. H. Lewy called attention to the fact that the basic factor common to most, if not all, neuropathies, whether they are due to diabetes; pregnancy; alcoholism; hunger; cachexia; pernicious anemia; poisoning with heavy metals, such as lead, mercury, manganese, bismuth, antimony or chromium; metalloid poisoning with arsenic or phosphorus; poisoning with carbon disulfide; infectious diseases, such as yellow fever, typhus, typhoid and paratyphoid, diphtheria, tuberculosis and leprosy, or some constitutional hereditary conditions, is probably a lack of vitamin B.[1] Wechsler substituted the term neuropathy for neuritis, since the histologic changes observed in all these metabolic, toxic and infectious conditions are not those of exudative inflammatory leukocytic infiltration but are the characteristic cloudy swelling and ballooning of the medullary sheath, coil formation of the axis-cylinder and finally granular disintegration, which are also observed in experimental deprivation of vitamin B_1. The process is one of a failure of normal restoration and repair rather than of inflammation.

Since vitamin B is an essential factor in the metabolic chain by which carbohydrates are made available for the complete oxidation of fats, lack of this vitamin ultimately results in acidosis. Lewy stated that the pathologic changes in the nerves may be considered a peptinization, probably resulting from this increasing acidification. In general, the disturbance of normal function may be summarized as follows:

The liver, which is the main storehouse of vitamin B, is affected by the various noxious agents associated with the metabolic, toxic and infectious conditions mentioned and is rendered unfit for storage purposes. When for any reason the mobile supply of vitamin B in the liver is depleted by one third, the fixed supply in the nervous system is drawn on, and symptoms appear. Four fifths of the supply of vitamin B in the liver can be depleted in one week on a diet free of that substance. Thus the importance of a constant and adequate supply of vitamin B becomes apparent.

Read before the Eye and Ear Section of the California Medical Association, Del Monte, May 2, 1939.

1. Lewy used the term vitamin B to designate the vitamin B complex and did not differentiate between the various elements in his discussion.

When the average civilized diet is considered in the light of its vitamin B_1 content, it suggests a deliberate conspiracy to create a lack of this vitamin. To give a rough idea of the situation, I shall cite Cowgill's figures comparing the vitamin B_1 contents of various foods. The value of 1 mg. of synthetic thiamin chloride being regarded as 1,000, which is considered an adequate daily intake under ordinary conditions, polished rice rates 1.6, unpolished rice 20, white bread 2, whole wheat bread 20, refined cane sugar 0.00 and cane molasses 150. Thus it may be seen that the use of white flour, polished rice and refined cane sugar deliberately removes much of the vitamin B_1 from its richest natural sources in the diet. With the exception of liver, a few varieties of nuts, wheat germ and yeast, there are no other foods rich in this most essential element. It becomes apparent that the likelihood of a deficiency of vitamin B_1 is not as remote as might be imagined, and an explanation for many vague pains and aches, as well as stubborn attacks of severe neuralgia, emerges.

Lewy called attention to the fact that loss of hearing, nystagmus and visual symptoms suggestive of retrobulbar neuropathy are common in many of the diseases which have been enumerated. As these symptoms are a frequently baffling occurrence in the experience of every specialist in ophthalmology and otology, the importance of this new development in therapy is obvious. Since my interest was awakened in this subject by my own experience, I find that much significant and valuable work, which has not yet been reported in the literature, is being done by a number of physicians. Borsook, of the California Institute of Technology, is getting excellent results in cases of tic douloureux with intravenous administration of thiamin chloride. Dr. Guggenheim, of Los Angeles, is soon to report his results in the treatment of nerve deafness with thiamin chloride. As with sulfanilamide, every day seems to reveal new indications for the use of thiamin chloride.

REPORT OF A CASE

The patient was referred to me in April 1938 because of severe bilateral tinnitus, which had begun three weeks previously, about three days after the last of a series of twenty-eight daily roentgen treatments of 303 r each and one exposure to radium of 5,760 milligram hours for an epidermoid carcinoma of the cervix uteri. The onset of the tinnitus was accompanied by severe pain in the cervical and upper lumbar regions of the back and a bandlike headache above the eyes and ears. Some impairment of hearing also was noted. The tinnitus in the right ear was described as being of three separate types, a screeching noise, a scraping noise and a pulsating noise synchronous with the heart beat. The tinnitus in the left ear was more intense than that in the right ear and was described as being like a fog horn, which persisted for a varying number of seconds, stopped for a second and then started again.

Examination revealed both tympanic membranes normal except for some moderate retraction of the left drum. Both eustachian tubes were readily politzerized, without any noticeable effect on the tinnitus. The hearing was charted on the Maico audiometer and an impairment which was most marked in the right ear and in the higher frequencies noted.

The patient had been unable to get a night's sleep with the help of a 6 grain (0.39 Gm.) dose of sodium amytal and was threatening to jump off the pier unless she got relief. The responsibility was mine. I must admit, that in the face of my

previous experience with severe tinnitus not referable to a disturbance in the middle ear, I was not optimistic about my ability to help her.

I recalled, however, a report given by Charles Martin at the Congress of Radiology in Chicago in 1937 concerning the treatment of radiation sickness with vitamin B and also some work that has been done in the relief of alcoholic polyneuritis by Strauss, Wechsler, Joliffe and Colbert. If the condition were neuritis of the eighth nerve resulting from the toxic absorption following the destruction produced by the extensive radiation, perhaps similar therapy would be of value. It was worth a trial, especially in the absence of any other promising form of treatment.

The treatment was therefore started with a daily dose of 10 mg. of thiamin chloride administered intravenously for two days, without any noticeable effect. The dose was raised to 15 mg. on the third and fourth days, and on the fifth day a marked decrease in the tinnitus was first noted. The daily dose of 15 mg. was continued until the ninth day, at which time the tinnitus had stopped in the left ear but still persisted in the right. The following day it was gone in both ears, and it has never recurred since. The audiogram showed a 5 to 40 decibel improvement in hearing, which may possibly have been due either to the absence of the masking effect of the tinnitus or to an actual improvement in the condition of the eighth nerve. There were no subjective symptoms of vestibular involvement. It is not impossible that the normal recovery from the radiation sickness might have played some part in the picture. However, in view of the experience of others with this type of treatment and the good theoretic background for the belief that lack of vitamin B_1 is common to many, if not most, neuropathies, I believe that the intravenous administration of thiamin chloride is a safe and logical method of dealing with similar manifestations in the field of ophthalmology and otology, and I recommend it to the attention of practitioners of these specialties.

Parenteral administration is desirable in order to produce a rapid saturation and because a neuropathy of the vagus nerve, according to M. Bielschowsky, may have resulted in inactivity and even atrophy of the glands of the gastric mucosa, interfering with normal digestion and absorption from the gastrointestinal tract.

Thiamin chloride may be given subcutaneously, intramuscularly, intravenously or even intrathecally with a wide margin of safety, doses of from twenty-five to fifty thousand times the average daily requirement being necessary to produce lethal effects.

An adequate supply in the diet should be assured at the end of the parenteral treatment to prevent a recurrence of the symptoms. This varies from approximately 50 international units for children to 300 for adults, 1 mg. of thiamin chloride being the equivalent of 330 international units.

SUMMARY

1. A case of severe bilateral tinnitus and impairment of hearing following extensive radiation therapy for carcinoma of the cervix uteri is reported in which relief was obtained by daily intravenous injection of thiamin chloride in 10 and 15 mg. doses for nine days.

2. Intravenous administration of thiamin chloride in one or more daily doses of 10 mg. for all neuropathies of the cranial nerves is suggested and its possible mode of action indicated.

110 Pine Avenue.

Abstracts from Current Literature

Ear

OTITIS MEDIA FROM THE PAEDIATRICIAN'S VIEWPOINT. G. A. CAMPBELL, Canad. M. A. J. **40**:146 (Feb.) 1939.

Campbell advises local treatment with chlorbutanol, ephedrine, glycerin and water for twenty-four to forty-eight hours in the average case of otitis media. If improvement has not been observed by that time, he advises myringotomy.

If the condition is in the acute stage, sulfanilamide is used and has been found effective in shortening the course of otitis and preventing complications involving the mastoid. A concentration of 4 mg. per hundred cubic centimeters of blood is maintained.

Toxic symptoms are rare.

During 1936 and 1937 the average number of days of hospitalization in cases of otitis was twenty-four and fifteen hundredths and the incidence of complications 8.2 per cent. During 1937 and 1938, with routine use of sulfanilamide, the average duration of hospitalization was reduced to eight and seven hundredths days and the incidence of complications to 4.1 per cent.

Campbell stresses the importance of latent mastoiditis as a cause of fatal marasmus; 53 per cent of all children who died in the hospital showed purulent mastoiditis at autopsy.

MOORE, Omaha. [AM. J. DIS. CHILD.]

SULFANILAMIDE IN THE TREATMENT OF OTITIS MEDIA. GILBERT E. FISHER, J. A. M. A. **112**:2271 (June 3) 1939.

Fisher reports striking results of the use of sulfanilamide in the treatment of acute otitis media due to the beta hemolytic streptococcus. In 95 cases sulfanilamide was not given, while in 88 cases it was given with an equal amount of sodium bicarbonate. In all these cases the red protruding drum membrane was incised, or, when spontaneous perforation took place, incision to enlarge the opening was performed in order to promote drainage. In the study, as arranged in tabular formation, it is seen that the numbers of patients receiving sulfanilamide and of those not receiving it were about equal for each month. No seasonal variation or difference due to the virulence of the infecting organism is apparent in this series. The months of December, January, February and March showed a larger incidence of aural suppurations.

The actual difference in treatment in the cases in this series is merely the use of sulfanilamide in one group and not in the other, and because of the care with which the study was conducted the results speak emphatically for themselves.

Of the 95 patients who had myringotomy but were not given sulfanilamide, 66 required mastoidectomy, whereas of the 88 patients who had myringotomy and were given sulfanilamide, only 7 required mastoidectomy. Four of the group not receiving sulfanilamide had septicemia; 3 of these recovered after operation, and 1 died.

No complications arose in the group of 88 patients who received sulfanilamide. The average duration of otorrhea for 95 patients who did not receive sulfanilamide was sixty-five days, while the otorrhea of the 88 patients who were given sulfanilamide lasted on the average twenty-three days.

The author has found 10 grains (0.65 Gm.) of the drug (with an equal part of sodium bicarbonate) three times daily for the first four days and 5 grains (0.32 Gm.) three times daily thereafter safe and effective.

The toxic symptoms that should be looked for and the undesirable effects of the drug are as follows: (1) increasing weakness and lassitude; (2) loss of appetite; (3) nausea and vomiting; (4) fever, which decreases when the drug is withdrawn and increases when its administration is begun again; (5) a progressive fall in hemoglobin; (6) dermatitis, especially of the exfoliative type; (7) severe hemolytic anemia with jaundice, at the appearance of which administration of the drug should be discontinued and a transfusion given, and (8) agranulocytosis.

When any of the first four symptoms appear, administration of the drug should be reduced or discontinued entirely. When any of the last four symptoms appear, administration should be discontinued at once.

GORDON, Philadelphia.

THE DEAFNESS OF VAN DER HOEVE'S SYNDROME IS DEPENDENT ON STAPEDOVESTIBULAR ANKYLOSIS. P. MOUNIER-KUHN, Oto-rhino-laryng. internat. **23**: 57 (Feb.) 1939.

This article is concerned with the otologic examination of a father and daughter with a heredity of van der Hoeve's syndrome. In both, the examinations revealed a negative reaction to the Rinne test, prolonged perception in the Schwabach test, lateralization of sound to the most affected ear in the Weber test, paracousis, normal reactions to the Gellé and Jacod tests and raising of the lower tone limit. The drum membrane was rosy and hypermobile, and the patients suffered with continuous tinnitus. Thus, the diagnosis was clearly otospongiosis.

It cannot at present be assumed, however, that the two diseases are identical; it can only be said that they are akin.

DENNIS, San Diego, Calif.

ASYMPTOMATIC THROMBOSIS OF THE LATERAL SINUS OF OTITIC ORIGIN. C. CALABRESI, Arch. ital. di otol. **51**:129 (March) 1939.

Calabresi reports on 2 patients who had thrombosis of the lateral sinus without clinical symptoms. One had subacute otitis; the other, chronic purulent otitis. In the case of the first the thrombosis was discovered in the course of a mastoidectomy, and as it was assumed from the appearance of the sinal wall that it was a matter of aseptic thrombus, the sinus was not opened. Recovery was uneventful. The second patient did not present symptoms of thrombosis. During the course of a radical exenteration of the mastoid the sinus was uncovered; the wall appearing normal, it was not opened. The patient died within forty-eight hours after operation in a state of cardiac collapse. Autopsy revealed an occluding thrombus in the horizontal portion of the sinus, extending to the beginning of the opposite sinus and the longitudinal sinus. The descending portion contained a parietal clot. Two small openings were observed in the wall of the vessel, and death was attributed to a large subdural hemorrhage. A low grade of microbic virulence was believed to be the cause of the latent progress of the disease.

DENNIS, San Diego, Calif.

CHRONIC SUPPURATION OF THE MIDDLE EAR AND VITAMIN C. F. CHIMANI, Monatschr. f. Ohrenh. **73**:123 (Feb.) 1939.

The author found a deficiency of vitamin C in cases of chronic suppuration of the middle ear. Consequently he administered vitamin C in such cases and improved the chronic otitis. Experimental work on guinea pigs revealed that vitamin C could be found in the mucous membrane of the middle ear but not in the mucosa of the nose and the mouth in animals fed with it.

LEDERER, Chicago.

A New View Concerning the Diminution of Hearing in Otosclerosis and Possibly a New Conception of Treatment. M. Meyer, Monatschr. f. Ohrenh. **73**:140 (Feb.) 1939.

The typical conduction deafness in otosclerosis is produced not only by fixation of the stapes but also by an increase in pressure within the spaces of the inner ear. Consequently decompression of these spaces by fenestration of the internal ear may be expected to improve the hearing. Meyer attempts to accomplish the same effect by suboccipital (cisternal) puncture. In fact, he succeeded with that kind of treatment in 11 cases. However, it is not possible as yet to say whether or not the effect can be considered permanent. He further calls attention to the fact that the diminution of hearing of high tones in patients with otosclerosis does not indicate degeneration of the cochlea, as in such cases high tones are well perceived by bone conduction.

Lederer, Chicago.

The Therapeutic Results in Thirty Cases of Otogenous Sepsis. D. Joannovich, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:17 (March) 1939.

In an eight year study of 556 cases of disease of the middle ear, the author observed and analyzed 30 cases of otogenous sepsis. In 18 of these the condition occurred in the course of acute otitis media, and in 12 it followed an acute exacerbation of chronic otitis media. He observed 23 cases of thrombophlebitis, and he noted intracranial complications in 12 cases. The most common organism was the streptococcus (21 cases), with the pneumococcus second (7 cases).

The usual treatment in such cases consisted of operation and removal of the focus of infection, ligation of the jugular vein, serum therapy, blood transfusions, chemotherapy and administration of vaccines. He performed ligation in 28 cases, in which he had 18 cures and 10 deaths. The vein was not ligated in 2 cases, in 1 of which the patient died. In 12 of the 21 cases of streptococcic infection treatment with Vincent's antistreptococcus serum was instituted. In these, he had 11 cures and 1 death. In the remaining 9 cases, there were 5 deaths and 4 cures. In the 7 cases of pneumococcic infection, he had 4 deaths and 3 cures, and he concludes that the pneumococcic infection always presents a much graver prognosis.

In view of the favorable results with Vincent's antistreptococcus serum, he suggests that it be recommended routinely in all cases of streptococcic infection. It can be given intramuscularly, subcutaneously or intravenously. The intravenous doses averaged from 15 to 60 cc., the intramuscular from 20 to 100 cc. and the subcutaneous injection from 5 to 60 cc. The maximum daily dose was 180 cc. and the minimum 30 cc.

In conclusion, he repeats that in 30 cases of operative otogenous sepsis he had 19 cures and 11 deaths, and he stresses the value of combining antistreptococcus serum with the usual therapy.

Persky, Philadelphia.

Transitory Vestibular Disturbances, with a Consideration of the Use of Frensel's Glass. J. Ohm, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:22 (March) 1939.

The author presented the case of a patient with transient vertigo of four days' duration and vomiting for the last two days.

Observations at general examination were essentially negative except for nystagmus, which the author measured with a nystagmograph. He noted that the nystagmus was weaker in a bright light, and hence he darkened the room, and in addition he used Frensel's glasses. By these procedures, he was able to determine that the patient had a lesion on the left side, either in the nerve or in the nucleus.

In conclusion, he suggests that the nystagmograph be used in all cases, particularly in those in which there are either transient or fine ocular movements, which are difficult to perceive or elicit. In addition, he suggested the use of a dark room and the Frensel glasses to aid in the determination of these minimal or evanescent signs.

Persky, Philadelphia.

ANALYSIS OF SIXTY-ONE CASES OF THROMBOSIS OF THE LATERAL SINUS OF OTITIC ORIGIN OBSERVED DURING FIFTEEN YEARS AT THE IMPERIAL UNIVERSITY HOSPITAL, OKAYAMA. SHIGERO TAKAHARA, SADAKUNI OKA and KINJIRO OHNO, Ztschr. f. oto-rhino-laryng. **45**:42 (Jan.) 1939.

A comparative study of the mortality in cases observed during 1921 to 1931 and in those observed during 1931 to 1936 is given.

1. There were 61 cases of sinus thrombosis during the fifteen years.

2. One thousand, five hundred and seventy-nine mastoidectomies were performed, an incidence of 3.9 per cent of thrombosis of the lateral sinus.

3. Other intracranial complications were meningitis in 60 cases and abscess of the brain in 15.

4. In 42 of the 61 cases, or 68 per cent, the patient made a full recovery.

5. Thirty-seven patients were male and 24 female; 12 were under 15 years of age, 30 between 15 and 50 years, and 18 above 50.

6. In 42 of the cases the condition originated from acute infection, in 28 of which, or 66 per cent, the patient recovered, and in 19 it originated from chronic infection, in 14 of which, or 73.6 per cent, the patient recovered.

7. The condition affected the right ear in 32 cases (chronic in 7 and acute in 25) and the left ear in 29 (chronic in 12 and acute in 17).

8. Metastasis occurred in 11 cases, in 4 of which the patient recovered.

9. The site of thrombosis was at the sinus transversus in 38 cases, at the sinus transversus and the bulb in 9 cases, at the bulb and the internal jugular vein in 1 case and at the sinus transversus, the jugular bulb and the internal jugular vein in 11 cases.

10. During the last five years the rate of recovery was 75.8 per cent, an increase from the 62.5 per cent of the previous ten years.

The authors attribute their success to the following measures:

1. Employment of Queckenstedt's test for early recognition of thrombosis. They consider that the difference of 50 mm. in the pressure of the spinal fluid between the two ears is indicative of the involvement of the venous channel.

2. Early ligation of the internal jugular vein when any of the following indications is noted:

(*a*) Signs of metastasis.
(*b*) Extention of the thrombosis into the internal jugular vein.
(*c*) Failure of organization of the thrombus in the lateral sinus or in the bulb.
(*d*) Persistent septic temperature after closure of the lateral sinus.

HARA, Los Angeles.

CONTINUOUS IRRIGATION OF THE SPINAL FLUID FOR MENINGITIS OF OTITIC ORIGIN. KIYOSHI SHIMIZU, Ztschr. f. oto-rhino-laryng. **45**:381 (March) 1939.

In a 10 year old boy acute meningitis developed as a complication of chronic mastoiditis of several years' duration. Study of the spinal fluid indicated strongly positive Nonne-Apelt and Pandy reactions. There were 2,201 cells, mostly polymorphonuclear, though culture did not show growth and Queckenstedt's sign also was negative.

On the evening of admission to the hospital radical mastoidectomy was performed. This was followed by continuous irrigation of the spinal fluid. For hydration the patient received 700 cc. daily of Locke's solution, both subcutaneously and intravenously, in addition to methenamine, 4 Gm. daily, in the vein. After this treatment the signs of meningeal irritation gradually subsided, and on the seventeenth postoperative day spinal irrigation was suspended. With the reappearance of untoward signs the irrigation was reestablished and continued until the cell counts became normal, which occurred on the fifty-seventh postoperative day.

The patient was dismissed on the seventy-third postoperative day. The irrigations were interrupted twice, for five and for three days, during the observation. The average amount of spinal fluid removed daily was 312 cc., the total in forty-six days 14,350 cc.

HARA, Los Angeles.

Pharynx

TONSILLECTOMY UNDER INTRAVENOUS ANAESTHESIA IN CHILDREN SUFFERING FROM CHRONIC RESPIRATORY DISEASES. K. HUTCHISON, H. S. MITCHELL and H. McHUGH, Canad. M. A. J. **39**:237 (Sept.) 1938.

Twenty-two cases are reported in which intravenous anesthesia was used. Pentothal sodium was used in the first group of cases and evipal in the later, larger group. In a few cases of the latter group morphine was used preoperatively.

The average dose of pentothal sodium employed amounted to 0.608 Gm., while of evipal 0.614 Gm. was employed. The average age of the patients was 8.9 years; the youngest patient was 3 years old and the oldest 16.

Intravenous anesthesia may be of value in selected cases, but the authors do not feel that it should displace the ordinary methods for healthy children.

MOORE, Omaha. [AM. J. DIS. CHILD.]

RETROPHARYNGEAL ABSCESS. C. W. EVATT, J. South Carolina M. A. **34**:287 (Nov.) 1938.

Retropharyngeal abscess may be acute or chronic. The chronic abscess is a tuberculous condition and usually is associated with Pott's disease or tuberculosis of the cervical glands. It is not discussed in this paper. Acute retropharyngeal abscess is usually caused by a streptococcus. At Roper Hospital in Charleston, S. C., the author reviewed cases of this disease in 4 adults and 8 children (3 under 1 year of age). The incidence was distributed equally between the sexes. The onset may be slow or rapid and is associated with tonsillitis. The condition should be diagnosed early and treated immediately with incision. It is best to have a tracheotomy outfit ready for use if necessary.

WESTON, Columbia, S. C. [AM. J. DIS. CHILD.]

BIOLOGIC SIGNIFICANCE OF THE TONSILS AND ADENOIDS AND OTHER EXTERNAL LYMPHOID MASSES. P. W. LEATHART, Brit. M. J. **2**:835 (Oct. 22) 1938.

This is a discussion along the lines indicated in the title. In general, it is a plea for conservatism in respect to the tonsils and adenoids. Leathart concludes:

"1. That the function of an external lymphoid mass is to collect micro-organisms from food or air and to grow those to which an immunity is needed.

"2. That the condition 'enlarged tonsils' alone is definitely an indication against their removal.

"3. That the adenoid facies is more commonly caused by catarrhal sinusitis than by an adenoid mass.

"4. That catarrhal sinusitis can be treated successfully in a large number of cases without operation, by giving a 'nasal aperient.' [This term refers to a relatively prolonged administration of potassium iodide.—Abstracter.]

"5. That tonsils and adenoids at the present time are removed too frequently and without due consideration of their important biological function.

"6. That many cases of rheumatic fever and acute nephritis that are going downhill can be cured or greatly benefited by removal of the 'carrier organ.'"

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

HAEMORRHAGE FOLLOWING TONSILLECTOMY. D. W. ASHCRAFT, Brit. M. J. **2**:1078 (Nov. 26) 1938.

The subject is treated under the three following headings:

I. Factors influencing the occurrence of hemorrhage following tonsillectomy: (*a*) preoperative; (*b*) operative; (*c*) postoperative.

II. Types of postoperative hemorrhage.

III. The management of hemorrhage following tonsillectomy.

The preoperative factors influencing the occurrence of hemorrhage following tonsillectomy are as follows:

1. Severe tonsillitis or peritonsillar abscess. Recent infections increase the risk of hemorrhages. Therefore, after the occurrence of one of these conditions, a few weeks should elapse before operation.

2. Infections of the nose or nasal accessory sinuses. Acute sinusitis is a contraindication to immediate performance of the operation, as is the common cold. Chronic sinusitis should not be overlooked. Treatment for this condition should be instituted before tonsillectomy is considered.

3. Bleeding. If there is a history of susceptibility to prolonged bleeding, an investigation should be made before operation. The common coagulation time is four to seven minutes; the bleeding time one to four minutes. If they are prolonged, calcium and alkalis should be given or a blood transfusion performed. If bleeding cannot be controlled, tonsillectomy, unless absolutely necessary, should not be considered.

4. Menstruation. The objection to an operation in this period is based on psychologic considerations rather than on fear of hemorrhage.

5. Jaundice. Tonsillectomy is contraindicated in the presence of jaundice.

6. Anemia. Tonsillectomy should not be performed unless preliminary treatment is given; even a blood transfusion may be advisable.

7. Type of patient. This concerns the adult rather than the child.

The operative factors are now taken up. The type of operation is discussed. Chloroform anesthesia or deep anesthesia induced by any other agent results in a fall of blood pressure. Therefore, when the blood pressure regains its normal level, hemorrhage may occur. Clot formation is liable to be imperfect in the larger veins in the bed of the tonsillar fossa. Especially is this likely to be so when there has been a complete division of a large longitudinal vein. The bleeding point must be searched for and tied. The maintenance of a clear airway is important after tonsillectomy. Hence it is important that an artificial airway be inserted in the mouth after all the blood clot has been removed. One must be certain that all ligatures are securely tied. Vasoconstrictors are not to be recommended for use at the time of operation because of the subsequent reaction of capillary ooze.

The postoperative factors are considered as follows. All conditions tending to cause congestion are to be avoided after the operation. In bed, the patient should be in the "lateral" position. The artificial airway should be removed when the cough reflex has returned. Restlessness after operation calls for a sedative. Children should have an enema containing bromides. No hot fluids are to be permitted during the first twenty-four hours. Cold fluids or semisolid food are given during the first day. The patient should be allowed out of bed for the first time on the evening of the third day following operation. No hot baths are allowed in the first ten days after the operation. For a week after leaving the hospital there must be avoidance of all strenuous exercise. A mixture containing potassium chlorate, sodium salicylate and sodium bicarbonate should be given three times daily throughout convalescence.

The types of hemorrhage are: (1) reactionary hemorrhage occurring within the first twenty-four hours after operation, (2) hemorrhage during convalescence and (3) true secondary hemorrhage. The management of these types is as follows: For the reactionary ooze the author prefers morphine in the case of adults and

children over 10 years but bromide and chloral hydrate for children under 10 years. The material on the treatment of reactionary hemorrhage is too extensive to abstract. Hemorrhage during convalescence occurs usually on the fifth night after operation and is due to factors already enumerated. As a rule, it is not severe, and when it is not the author uses a gargle of weak hydrogen peroxide. The clot should not be removed, as it may remove itself after the act of gargling. True secondary hemorrhage is much more serious than the other types. It is also rare, Control of such hemorrhage is difficult in many cases, as it is seldom possible to clamp and ligate a single bleeding point. Recourse to suturing the faucial pillars may be necessary. Other measures which have been recommended are of doubtful value according to the author's experience.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

Larynx

ACUTE LARYNGITIS IN INFANTS. G. J. GREENWOOD, Illinois M. J. **75**:52 (Jan.) 1939.

Following a review of the anatomy, histology, bacteriology, symptomatology and therapeutics of acute laryngitis in infants, with a discussion of 14 cases observed at the Children's Memorial Hospital during the winter of 1933-1934, Greenwood arrives at the following conclusions: Specific laryngitis in infants is a pertinent pediatric problem. When signs of obstructive dyspnea appear, the endoscopist or the endoscopically trained laryngologist should be summoned. For immediate relief intubation or tracheotomy is necessary. Greenwood believes that in an infant low tracheotomy conserves the laryngeal structure better than intubation.

BARBOUR, Peoria, Ill. [AM. J. DIS. CHILD.]

CHORDECTOMY BY MEANS OF SURGICAL DIATHERMY. T. DEMETRIADES, Monatschr. f. Ohrenh. **73**:119 (Feb.) 1939.

The writer removes tumors of the vocal cords by means of the electrical knife. The advantage of this procedure is supposed to be the avoidance of postoperative hemorrhages.

LEDERER, Chicago.

THE PATHOLOGIC HISTOLOGY OF LARYNGEAL TUBERCULOSIS. H. ESCHWEILER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:31 (March) 1939.

The author discusses the different modes of tuberculous infection of the larynx and presents sections from cases wherein the hematogenous route may be a factor. He believes that primary infection of the larynx is rare, even in cases in which a pulmonary lesion is not demonstrable. In discussing the involvement of the regional lymph glands, he states that it varies in degree in laryngeal tuberculosis, because of the paucity of the lymphatic system about the intrinsic larynx, where one finds but few glands, while, on the other hand, an infection of that portion of the larynx which is richly supplied by a lymphatic system, such as the introitus of the larynx and the sinus piriformis, can be drained by the glands in the membrana hyothyreoidea and carried to the superior cervical glands and then to those about the common facial vein and the internal jugular vein. However, in cases of the latter type, one must be careful to eliminate a descending infection from the tonsils. The author has also presented a specimen in which he was able to trace a tuberculous infection from the entrance of the larynx and the sinus piriformis to the glands and the membrana hyothyreoidea.

In the incipient stage of the infection there is a marked exudative reaction and a distortion of the involved structures, with a subsequent marked invasion of the lymphatic system. However, in a secondary or later stage, immunity is established in the lymphatic system. In the tertiary stage, there is often evidence of an exudative and a productive process. The author believes that the edema of

the larynx is really a stasis, particularly in a chronic form. In the acute form, he states, the edema may result from some toxic influences on the walls of the blood vessels.

In conclusion, he believes that the symptoms must be carefully correlated with the histologic picture for an accurate evaluation of the underlying process.

PERSKY, Philadelphia.

Nose

OSTEOMYELITIS OF THE SPHENOID BONE—REPORT OF FIVE CASES WITH THE AUTOPSY FINDINGS. HOWARD C. BALLENGER, Ann. Otol., Rhin. & Laryng. **48**:95 (March) 1939.

Ballenger discusses, briefly, the reports of osteomyelitis of the sphenoid bone and mentions that sphenoid sinusitis is responsible for about 35 per cent of all intracranial complications of nasal origin. The spread of the infection to the meninges was found to be predominantly vascular, particularly via the sphenoid mucosa and through the vascular marrow spaces of the sphenoid. He feels that in at least a vast majority of cases there must of necessity have been some form of osteomyelitis. The etiologic factor in most cases seems to have been preceding sphenoiditis, although the possibility of retrograde thrombophlebitis from the cavernous sinus or an extension from petrositis must be borne in mind. Rarely thrombi or bacteria from a distant focus may lodge in the vascular spaces of the sphenoid. Diagnosis of osteomyelitis of the sphenoid before the occurrence of intracranial complications is difficult. The usual signs of sphenoiditis are usually present. Signs of sepsis appear as bone is involved, but localizing signs are absent. Roentgen examination may show a cloudy or vague outline of the sinal wall. This increased density may extend into the middle fossa or along the base of the skull. The sella may show changes associated with osteitis or osteomyelitis. Pfhaler says that the sella may show calcification of the bridge or ligaments connecting the anterior and posterior clinoid processes. Calcification, or a flocculent deposit of lime, may be seen in the bands behind the posterior processes. The organisms usually found are Pneumococcus type III and the hemolytic streptococcus. The most frequent complication of osteomyelitis of the sphenoid is meningitis and next thrombosis of the cavernous sinus. He reports 5 cases of osteomyelitis of the sphenoid.

M. V. MILLER, Philadelphia.

THE TREATMENT OF RHINITIS CHRONICA HYPERPLASTICA. BAER, Monatschr. f. Ohrenh. **73**:108 (Feb.) 1939.

The writer uses endonasal instillations of 50 to 60 per cent solutions of dextrose for the treatment of rhinitis chronica hyperplastica and reports good results.

LEDERER, Chicago.

AN INSTRUMENT FOR THE OPENING OF THE MAXILLARY SINUS THROUGH THE INFERIOR MEATUS. W. HAARDT, Monatschr. f. Ohrenh. **73**:110 (Feb.) 1939.

The author has constructed a new instrument for the opening of the maxillary sinus through the inferior meatus. As he does not illustrate the instrument it is difficult to judge its practical importance and originality. LEDERER, Chicago.

A CONSIDERATION OF THE RADICAL OPERATION ON THE FRONTAL SINUS (SEIFFERT'S MODIFICATION). K. WÜRFEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:46 (March) 1939.

Würfel discusses various types of radical operations on the frontal sinus—Kuhnt's, Jansen's, Riedel's and Killian's—and he also outlines their limitations. In view of these, he recommends as an operation of choice the Seiffert modification of the Killian operation.

His technic is as follows: After the completion of the Killian operation and just prior to the suturing of the skin, he makes sure that there is a wide nasofrontal communication. He inserts a rubber-covered tampon through the nose into the frontal sinus. Along with this tampon is usually a small drainage tube to take care of any mild drainage from the sinus. At the upper end of this tampon, he has previously placed a layer of Thiersch skin graft. This flap extends into the frontal sinus, with its superior surface placed so that it covers the supraorbital ridge and approximates the suture line. The flap must be large enough to extend from the septum to the supraorbital ridge and then upward to the edge of the sinus. The outer skin is then sutured, and a pressure bandage is applied externally. Four to seven days after the operation, the nasal pack or tampon is removed.

The author analyzed a series of 136 cases in which operation was done by this method between the years 1923 and 1934. In this group, he was able to follow up 72 patients. The results were striking in that after five years he was able to maintain a widely patent nasofrontal opening; the resultant scar was scarcely noticeable; the external deformity was negligible, and, because of the thoroughness, there were few recurrences, which were due either to underlying ethmoiditis or to a recurrent infection of the upper part of the respiratory tract. In this series he had only 13 complications and 11 deaths. Among the complications were meningitis in 2 cases and osteomyelitis in 3.

He recommends this technic because it shortens the convalescence, reduces postoperative complications, minimizes the number of recurrences and maintains a widely patent nasofrontal duct for a long time. Failure of the graft to "take," with secondary necrosis and sloughing, occurred in only rare instances.

PERSKY, Philadelphia.

A SOLITARY OSTEOMA OF THE ETHMOID LABYRINTH EXTENDING INTO THE ORBIT. WILHELM SCHOLZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:86 (March) 1939.

A case of osteoma of the ethmoid labyrinth which extended into the orbit in a 16 year old patient is reported. The patient had not complained of any previous symptoms referable to the nose or throat, but had noticed that for the past two to three years the eyes were slowly distorted outward and forward. Pain, disturbance of vision or interference with breathing did not occur.

Examination revealed a hard crescentic swelling on the nasal wall of the orbit. Roentgen examination revealed a massive shadow extending into the right orbit, irregular in outline but sharply defined, with a pedicle extending into the right ethmoid labyrinth.

At operation, the author found a hard white tumor, which was freely outlined and was attached to the ethmoid labyrinth by a short pedicle. He was able to remove the wall of the labyrinth without laceration of its mucous membrane. The patient recovered without any untoward events during convalescence.

Histologic examination showed the tumor to be a fibro-osteoma.

PERSKY, Philadelphia.

FIVE CASES OF EXOPHTHALMOS CAUSED BY ETHMOIDITIS. AKIRA NAKANO and KAZUO SATO, Oto-rhino-laryng. **12**:105 (Feb.) 1939.

The authors report 5 cases of ethmoiditis with symptoms referable to the eye, especially exophthalmos. They review the manner in which the pathologic changes occurred.

CASE 1.—A man aged 49 gave a history of chronic ethmoiditis on the right associated with frontal headache, double vision, photophobia and impairment of hearing and vision of about a year's duration. A Denker operation was followed by intranasal ethmoidectomy. Within a week after the operation the vision was restored, and the exophthalmos gradually disappeared.

CASE 2.—A woman 49 years old presented a strongly positive Wassermann reaction with a history of ethmoantritis on the left of seven years' duration. Three months previously she had had a cold associated with headache, impairment of vision and exophthalmos on the left. After transantral ethmoidectomy, the vision was greatly improved, and the exophthalmos completely disappeared.

CASE 3.—The patient was a boy 8 years old.

CASE 4.—In a woman 28 years old exophthalmos developed shortly after she had taken a cold. At the time of external ethmoidectomy, slight change other than hyperemia of the mucosa was noted in the ethmoid capsules. The exophthalmos disappeared after the operation.

CASE 5.—A man aged 35 had had a bilateral Caldwell-Luc operation twenty years before. About three years before coming under observation he noticed impairment of vision which was associated with exophthalmos on the right side which gradually became worse. At external ethmoidectomy a large mucocele was disclosed. Ten days after the operation, the vision was recovered, and the exophthalmos became almost unrecognizable. The article is well illustrated with photographs of the patients and roentgenograms of their paranasal sinuses.

HARA, Los Angeles.

Miscellaneous

ACUTE BULBAR POLIOMYELITIS FOLLOWING RECENT TONSILLECTOMY AND ADENOIDECTOMY. M. STILLERMAN and A. E. FISCHER, Am. J. Dis. Child. **56**:778 (Oct.) 1938.

Stillerman and Fischer studied the 686 patients with poliomyelitis who were admitted to the Willard Parker Hospital during 1935, in order to determine whether the removal of tonsils and adenoids in any way influenced the onset or clinical type of the disease. It was found that tonsillectomy and adenoidectomy had been performed in 10 cases during the month antedating the illness. In 8 of these cases poliomyelitis began from ten to twelve days, and in the other 2 cases, sixteen and twenty-two days, respectively, after the operation. Six of 10 patients had the bulbar or encephalitic form of the disease. Careful inquiry was made among 52 patients with poliomyelitis who were admitted to the hospital during 1937. On 3 of these patients tonsillectomy had been performed from two to three weeks preceding the onset of poliomyelitis. These 3 children had the bulbar type of the disease. All of them died. The bulbar or encephalitic type of poliomyelitis was present in 68 of the 686 patients and in 15 of the 52 patients, while in 6 and 3 patients, respectively, who recently had had tonsils and adenoids removed the bulbar or encephalitic type developed. Thus, there was a disproportionately high percentage of bulbar involvement among the tonsillectomized patients in both the 1935 and the 1937 outbreak. This much higher incidence of bulboencephalitic forms suggests the operative field as the port of entry of the virus. The authors conclude, therefore, that tonsillectomy and adenoidectomy should not be encouraged during an epidemic of poliomyelitis.

AUTHORS' ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.].

CERTAIN THERAPEUTIC EFFECTS OF WHOLE SUPRARENAL GLAND BY MOUTH. O. E. BARBOUR, Arch. Pediat. **55**:661 (Nov.) 1938.

Over 380 patients are included in this study. Desiccated whole adrenal gland was administered to infants or children of the allergic or vagotonic type. The optimal dose seemed to be 1 grain (0.064 Gm.). If the conditions treated were acute, the substance was administered at two or three hour intervals.

One hundred and forty children with bronchial asthma were treated, and relief was obtained by 74 per cent. The adrenal gland seemed to assist in the cure of 32 of these children.

Forty-four patients with spasmodic croup were treated, and relief was obtained for 90 per cent.

Eighty infants suffered from pylorospasm; vomiting was relieved for 90 per cent. For those who did not improve within one or two days, adrenal gland was augmented with iodine or thyroid gland, a procedure which usually produced the desired results.

Eighty patients with eczema were treated, and adrenal gland was effective, alone or when administered with iodine or thyroid gland, in relieving 80 per cent. Improvement was prompt, usually occurring within one or two days. When the optimal dose was established and maintained, the skin remained free from the rash in 80 per cent of the patients. Food allergy presented no great problem when endocrine therapy was employed.

In treating 40 patients with hypothyroidism, adrenal substance proved most valuable when used in conjunction with thyroid substance. More adequate amounts of thyroid could be administered when adrenal material was administered than when thyroid was given alone.

For purposes of comparison and as partial controls, treatments other than that with desiccated adrenal gland were given alternately or concomitantly at intervals through the course of this study. Drugs which simulated the therapeutic action of adrenal gland were ephedrine and neosynephrin.

Iodine and atropine reacted to the adrenal therapy much as did thyroid gland. They appeared to inhibit the untoward reactions and at the same time to enhance the effectiveness of the therapy. In some patients untoward reactions occurred while adrenal gland was being administered, such as abdominal pain, vomiting and constipation.

ORR, Buffalo. [AM. J. DIS. CHILD.]

TREATMENT OF ACUTE INFECTIONS OF THE CENTRAL NERVOUS SYSTEMS WITH SULFANILAMIDE. J. B. NEAL, J. A. M. A. **111**:1353 (Oct. 8) 1938.

Neal reviews the mortality statistics of large series of cases of meningitis. The various virulent and bacterial types are mentioned, as well as the effect of sulfanilamide on each. The most remarkable change has been the reduction in the mortality from meningitis due to Streptococcus haemolyticus from over 95 per cent to 20 per cent in 27 cases. Some reduction occurred in the incidence of meningococcic meningitis; little, in that of pneumococcic meningitis and the other types. Drainage of the focus of infection is essential.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.]

SULFANILAMIDE IN TREATMENT OF BRAIN ABSCESS AND PREVENTION OF MENINGITIS. P. C. BUCY, J. A. M. A. **111**:1639 (Oct. 29) 1938.

Bucy reports a case in which an abscess of the brain was cured and a condition which was almost certainly meningitis due to hemolytic streptococci prevented by the administration of sulfanilamide. Owing to an error in diagnosis, a cerebellar abscess on the right was exposed with bilateral suboccipital craniectomy. The contents of the abscess were aspirated, and the surface of the cerebellum was contaminated with pus swarming with hemolytic streptococci, the ventricular system and the subarachnoid space being thus exposed to this infection. The abscess was not drained. The administration of sulfanilamide was begun at once. The patient improved steadily. There was almost no febrile reaction, and at no time did signs either of meningitis or of refilling of the abscess develop. The patient had almost completely recovered within two weeks.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

TOPOGRAPHIC SYNDROMES OF THE CENTRAL VESTIBULAR PATHWAYS IN MAN. J. A. BARRÉ, Rev. d'oto-neuro-opht. **16**:420 (June) 1938.

This article is a continuation of Barré's report to the Tenth Congrès des Sociétés d'Oto-Neuro-Ophtalmologie. The first part was published in the May 1937 issue of *Revue d'oto-neuro-ophtalmologie* (page 321).

In order to clear up misunderstandings of the terms "deficit" and "irritation," Barré defines them as follows: By deficit, as applied to a nerve pathway, is meant absence, disturbance or diminution of conduction and the disturbances resulting from it. For the vestibular apparatus, a deficit would be shown by disturbance or lack of sensation or reflex, in whole or in part, in the distribution of its components. The term irritation implies a redoubling of physiologic activity; this activity is abnormal because of its degree or duration. The phenomena of deficit and irritation may coexist in a large number of pathologic conditions. In certain other cases each may appear in a pure state. They are common to the central and the peripheral neurons. That a lesion may have a double effect is proved by the frequent observation of a case in which conduction is arrested in the peripheral neuron by a lesion that at the same time is the source of abnormal excitation of the central neuron. Certain lesions produce particularly the signs either of deficit or of irritation, so that clinical analysis may warrant an opinion concerning the nature of the lesion. An acoustic tumor, of slow development, may exist for years without producing symptoms other than unilateral deafness. Other lesions, developing more rapidly, produce early marked tinnitus, while the hearing is only slightly affected. Barré cites the vestibular disturbances observed in cases of syringobulbia and multiple sclerosis as examples of irritative syndromes. Complex syndromes are produced by lesions that involve both the vestibular apparatus and the cerebellum. Only recently has an attempt been made to analyze separately the syndromes of these two regions. Vestibular hypotonia associated with cerebellar hypotonia causes excessive nystagmus; in the test of the extended arms, however, it may happen that either one or both arms, which would be displaced horizontally in a given direction if the vestibular apparatus alone were involved, remain immobile or deviate to the opposite side if the cerebellum also is involved. The vestibular harmony has been broken by participation of the cerebellum.

The vestibular syndromes may be grouped into a few general types: complete, partial and associated and combined syndromes. The vestibular syndromes are made up of (1) subjective signs, such as sensation of vertigo or pulsion; (2) spontaneous or revealed objective signs (deviations of the head, eyes, trunk and arms or rotation around the axis), and (3) provoked objective signs (revealed by caloric, rotatory and galvanic tests). The complete vestibular syndrome is unilateral or bilateral. When it is bilateral, there may be predominance on one side; nystagmus or a deviation may be so accentuated on one side as more or less to mask the same element of the syndrome on the opposite side. In such a case, only instrumental tests will determine the respective value of one or the other side. Partial vestibular syndromes have been represented until recently by the partial vestibulo-ocular syndrome or the vestibulospinal syndrome. Of the partial vestibular syndromes an important example is crossed vestibular dysreflexia or areflexia, in which the causal lesion is in a restricted segment of the central vestibular pathways, a part of the chiasm of the vestibular pathways.

With a large number of lesions involving the peripheral and bulbomesencephalic segments of the vestibular tracts one observes besides the spontaneous or provoked reactional movements a modification of the general bodily equilibrium. With lesions situated in the temporal, parietal and frontal poles, disturbance of the axial equilibrium of the body is accentuated. Barré believes that the peripheromesencephalic vestibular apparatus is integrated into the general apparatus of equilibration and constitutes a reflex mechanism, the sensorial and motor pathways of which are articulated in the bulb and peduncle. Above it lies an apparatus

for automatic regulation, reduced to a small number of pathways that are assembled in the red nucleus and perhaps a little higher. The hemispheric cortex probably plays an important role in the perception of the sensations of equilibrium or disequilibrium; it is the conscious and voluntary mechanism, disturbance of which is so often shown by retrolateropulsions or other deviations of the axis of the body or even by falling. Ascending pathways from the otoliths, canals and posterior columns and perhaps other pathways are dissociated and distributed to various cortical areas on arriving at the subthalamic or retrothalamic region. That association pathways from these areas connect with the frontal or prefrontal cortex is probable. It is not astonishing that when the prefrontal area is affected directly or indirectly it may be the point of passage or departure of a disturbance in general equilibration. A special disturbance of equilibration will be created when the representation of space is altered; a different disturbance will occur when the faculty of attention is diminished.

A comparison of the clinical data with the anatomic arrangement is relatively easily made in cases of lesions of the vestibular nerve. This is more difficult with lesions of the bulb. The studies of Lorente de Nó indicate a greater complexity of the intrabulbar reflex pathways than had previously been supposed. Clinical observations indicate a precise anatomic localization for the lesion causing crossed dysreflexia, in one half of the pons, near the midline and slightly above the nucleus of the sixth nerve. This has as yet no demonstrable anatomicoclinical support. In the peduncular region superposition of the clinical and the anatomic descriptions is again easy. The schema of van Gehuchten and Winkler permits a clear understanding of the various particulars of the vestibular syndromes of lesions of the peduncular tegmen, whether they are situated below, at or a little above the red nucleus. Lesions of the hypothalamic cross roads produce diverse disturbances, but the number of cases of vestibular syndromes known to be referable to this region is not sufficient to determine whether the relation is explicable by its anatomic structure. In the regions located still higher the vestibular pathways have scarcely been mapped.

Dusser de Barenne and de Kleyn have shown that in rabbits after the ablation of one cerebral hemisphere the two vestibular mechanisms remain equally excitable and that the quick component of nystagmus, which is toward the side of operation, may be much stronger than that on the other side, whatever the method used to provoke it. The same phenomenon has been observed in man, either as simple predominance of the direction of nystagmus or in a pure and complete state. It is interesting that a minimal or even fugacious lesion in man can produce the same syndrome as ablation of an entire hemisphere in the rabbit. Muskens' experiments on animals led him to postulate "supravestibular pathways," which arrive at the pallidum via the posterior commissure. Serious doubts have been expressed about Muskens' interpretations, but he demonstrated that various *mouvements de manège* and axial rotation can be produced by operation on the highest part of the peduncles. The tendency to axial rotation observed in man in certain cases, associated with a homolateral or contralateral pulsion of the body, generally without nystagmus and deviation of the arms, is a new and important fact and directs attention to the anterior peduncular region. The experiments of Foerster, Spiegel, de Kleyn and Versteegh and Benjamin have yielded interesting data on the physiologic reactions of the region surrounding the interparietal sulcus. This is the only point at which excitation can produce the sensation of vertigo in man. It is probable that the mechanisms of the quick component of nystagmus and of the optokinetic reflex are in connection with this sulcus; it is also probable that fibers from the utricular otolith end there, while those from the saccular otolith go to the auditory center in the temporal lobe. Parietal lesions occupy the first rank among corticosubcortical lesions that may disturb general equilibration.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

INTRODUCTION TO THE STUDY OF VESTIBULAR DISHARMONY. I. ALFANDARY, Rev. d'oto-neuro-opht. **16**:577 (Nov.) 1938.

The vestibular apparatus occupies an important place in the semeiology of the nervous system. Examination of it is indispensable for the topographic diagnosis of lesions of the cerebral trunk, and the discovery by Barré of vestibular disharmony renders possible the diagnosis of cerebellar lesions, even in the absence of classic cerebellar symptoms. In a harmonious vestibular syndrome all reactions are directed toward the side of the labyrinth excited. However, in certain diseases of the cerebellum or of the brain stem the nystagmus (quick phase) is directed toward the side of the lesion. This phenomenon has been attributed to compression or inflammatory infiltration of the vestibular centers. These hypotheses are supported by experiments and clinical observations, which show that a low bulbar lesion produces homolateral nystagmus, while a lesion at the middle of the bulb causes contralateral nystagmus. Disharmony is a combination of homolateral and contralateral vestibular symptoms, to which is often added the spontaneous, incomplete syndrome: the absence of one of the elements of the vestibular syndrome. In the bulb, the vestibulospinal bundle at a certain level is separated from the vestibular nuclei, and one of them may be affected by a lesion and not the other. In the disharmonious vestibular syndrome the following may be observed: 1. The nystagmus is homolateral; the slow phase is directed toward the side opposite that of the deviation of the trunk and extremities. 2. The deviation of the trunk and extremities is toward the side opposite the irritation. 3. If there is deviation of the head, it is in a direction contrary to that of the other deviations. 4. Deviation during walking is in a direction contrary to the rule. 5. Subjective vertigo is in a direction contrary to the rule.

The author makes the following conclusions: 1. The pure vestibular syndrome is always harmonious, whether the lesion is central or peripheral. 2. The vestibular syndrome becomes disharmonious under the following conditions: (*a*) Lesions of the cerebellum involving the cerebellovestibular pathway; this path is direct and crossed, and its function is to coordinate reflex movements destined to maintain the equilibrium of the body. (*b*) Bulbar lesions, either lesion of the vestibulo-oculomotor pathway, which is crossed, or a bilateral lesion of the bulb. In the first instance only the nystagmus is disharmonious; in the second the deviations may also be disharmonious. It is probable that a lesion involving the juxtarestiform body, by disturbance of the same pathways, can produce the same syndrome as a lesion of the cerebellum. (*c*) Peduncular lesions, in which the disharmonious syndrome has not yet been satisfactorily explained. 3. The disharmonious syndrome always indicates a central lesion of the posterior fossa; most often, it indicates a cerebellar lesion. It must be considered a cerebellar symptom of the same value as other cerebellar symptoms of the series of Babinski and André-Thomas, with the reservations already indicated with regard to bulbar lesions.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

RESONANCE OF THE SKULL IN RELATION TO THE STATE OF THE LABYRINTH. T. DEMETRIADES, Rev. d'oto-neuro-opht. **16**:608 (Nov.) 1938.

While examining patients with intracranial tumors, Demetriades noticed that in the majority increased cranial resonance coincided with the side of the tumor and of the most marked labyrinthine lesion. Experiments demonstrated that the blood vessels of the cranium play an important role in cranial resonance, the latter being increased on the more hyperemic side. The resonance was measured by placing a band to which were attached two auscultation tubes, around the forehead and the two mastoid regions, setting a vibrating tuning fork on the forehead and comparing the resonance of the two sides of the head through the auscultation tubes. The resonance of the right side is normally greater than that of the left, supposedly because of the greater tonus of the right labyrinth. Other experiments

showed that the labyrinth (vestibular nerve) influences the vascular equilibrium, and hence the resonance, of the head. Stimulation of the labyrinth causes contraction of the vessels and reduction of the resonance of the side to which the nystagmus is directed. On the other hand, a lesion of a labyrinth causes a decrease in the resonance on the corresponding side of the head. Hence, the strongest resonance is found on the side of the most irritable labyrinth, the brain and the rest of the head being normal. If, now, sufficient encephalic hyperemia is produced to overcome the anemia caused by the labyrinthine lesion, the following syndrome appears: increased resonance on the side of the more feeble labyrinth. Examination in 27 cases of cerebral tumor revealed that the syndrome was present in 22. It is important to use Kobrak's caloric method, modified by Demetriades, in order to discover the finer shades of labyrinthine excitability. The phenomenon occurred in all cases of extensive hyperemia of the head, provided the latter or its cause produced a labyrinthine lesion. Such hyperemia accompanies cerebral tumors especially. Disease of the accessory nasal sinuses, malignant tumor of the epipharynx, otitis media and acute mastoiditis may influence cranial resonance.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

ISIDORE FRIESNER, M.D., *President, Presiding*

ABSTRACTED BY DR. ALFRED LEWY, CHICAGO

Seventy-Second Annual Meeting, Rye, N. Y., May 22-23, 1939

OTOLOGIC ADSPICE: PRESIDENT'S ADDRESS. DR. ISIDORE FRIESNER, New York.

The title is taken from the seal of the College of the City of New York, which presents a woman's head looking backward, "Respice"; outward, "Adspice," and forward, "Prospice." If one bases one's judgment soundly on the past and looks honestly at the present, one can view the future with hope and equanimity.

In every field a review of accomplishment, an evaluation of facts and a basis for their application are essential. In business this is called taking stock or inventory, perhaps to calculate pecuniary profit. In medicine the object is to prevent, ameliorate or cure the ills of others. This alone argues that medicine is not a business.

In discussing recent accomplishments in their field, otologists must include related ones in the general field of medicine and science.

Knowledge of the minute anatomy of the temporal bone has become more widespread, but it is still not known how one hears. The Wever and Bray phenomenon indicates that sound causes changes in the electrical potentials of the cochlea. Others show that these may arise from the membranes rather than the organ of Corti.

The Ménière syndrome due to a variety of causes basically seems to be linked to the metabolism of salt and water. On the other hand, if it is always due to interlabyrinthine changes why is the nerve section not always successful?

The advocation of new remedies is followed too often by indiscriminate application by enthusiasts. The successes are reported; later the unfavorable effects gradually become known. The emphasis of the published reports is laid, to paraphrase Carlisle, on "what the eye desires to see." Otologists are paying more attention to conservation of hearing. This is based on changes in their estimate of values. Formerly the advancement of a student was based almost entirely on scholastic achievements. Today more stress is laid on his ability to adjust to his environment—on his ability to meet life. Any handicap may cause a feeling of inferiority. Otologists are concerned with defects of hearing and disfigurements due to operative procedures. Restoration of a damaged facial nerve is in line with these efforts.

There is an effort for more conservative management of sinus thrombosis. Some are preferring the more horizontal incision in the neck for cosmetic reasons. Increasing experience has demonstrated that surgical intervention on the jugular vein is not invariably necessary in such cases. Sulfanilamide and its derivatives have helped in controlling bacteremia but have little or no effect on avascular tissues, so that the infected thrombus must still be dealt with surgically. There is a departure from the laissez aller attitude toward progressive deafness. The relation between loss of hearing and nutritional disturbances, especially vitamin deficiencies, is being studied; radiation treatment of adventitious lymphoid tissue,

especially about the eustachian tube, is used. But the greatest interest has been aroused by the surgical approach to restoration of hearing. To evaluate this therapy critically one must know the immediate perils due to technic and also the remote dangers. One must know the degree and duration of improvement and how it compares with that obtained by hearing aids. Until all the facts are known one should neither condemn nor exalt the new procedures.

In dealing with infections of the mastoid greater efforts are being made to conserve hearing. To quote Smith, "We remove the danger and allow as much of the conducting apparatus to remain as is consistent with safety." This applies also to deeper infections of the temporal bone.

Outstanding among the advances is the application of sulfanilamide and its derivatives as a bacteriostat not only against Streptococcus haemolyticus beta but against pneumococci in the form of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine). I cannot forego special mention of the successful treatment of meningitis by these drugs. Heretofore the isolated recoveries were considered miracles. For this one should be grateful.

I conclude that otologists may look to the future (Prospice) with confidence that their specialty will continue to occupy the honorable and important place which it deserves.

MODERN VIEWS REGARDING THE ANATOMY AND PHYSIOLOGY OF THE VESTIBULAR TRACTS. DR. OTTO MARBURG, New York.

(This article is essentially an anatomic description and does not lend itself to abstracting. Some of the high lights are touched on here.—A. L.)

The first question is whether the stimulation of the vestibular apparatus is an acceleration, a push or a static wave. In the lower fishes one has the lateral line system, an open channel, stimulated by the waves of the surrounding water. In higher animals there are added to the intermediate stage of a saccule and two canals a vestibule and three semicircular canals. The wave stimuli can be produced by turning, change in temperature or electricity and also by a blow or the vibration of the tuning fork. The nerves conduct the impulses, the nervus sacculoampullaris and the nervus utriculoampullaris comprising what I prefer to name the nervus labyrinthicus (vestibularis). I also reclassify the vestibular nuclei as nuclei labyrinthici pars intrafascicularis (ventrocaudalis), nuclei pars supra-fascicularis (triangularis), which he says are really one nucleus, and nuclei labyrinthici pars angularis (Bechterew). These constitute the real nucleus laby-rinthicus. These are accompanied by the small, cellular nucleus triangularis proper. There are also direct vestibular fibers which end in the oldest part of the cerebellum, the flocculus, the lingula and the nodulus. There are collaterals to Deiters' nucleus. It is possible that fibers or collaterals also reach the reticular nuclei. Every part of the labyrinth is connected with more than one nucleus. Most of the fibers cross by way of the internal arcuate fibers; those from the angular part cross partly at the posterior commissure. The courses of a number of other tracts are traced in detail to their connections with the nuclei of the muscles of the eye.

The oldest function of the labyrinth is the "skew deviation" (every change of position changes the position of the eye), which exists in the lowest fishes and corresponds to the righting reflex. The clonic, or quick, component of nystagmus indicates the side of the lesion in the pons and medulla. These movements are innervated by the healthy side of the labyrinthine nuclei with their crossed fibers. The slow component is to the healthy side because the tonic innervation is not crossed. (The fibers of the angular nucleus cross in the posterior commissure.) The conclusion is that the quick component depends on the pars fascicularis and its pathways, the slow component on the angular nucleus or the nucleus reticularis ventralis tegmenti pontis and their continuations. It appears, further, that the rotatory and horizontal movements are represented by the nuclei behind the sixth nerve. The upward and downward movements are still under discussion. I believe that the nuclei of the labyrinth and the posterior longitudinal bundle innervate the anterior oculomotor nuclei, perhaps supported by the nuclei for the righting reflexes.

Optokinetic nystagmus and cortical innervation of associated movements of the eyes also are traced. Dizziness I look on as a complicated symptom caused by different disturbances in the vestibular system. Conscious perception of vestibular impressions means only impression of direction. Influence of the vestibular on the vegetative system emanates from the proper triangular nucleus by way of the fasiculus triangulointercalatus into Roller's nucleus, ventrad from the hypoglossal nucleus.

To study the reflexes of the neck on labyrinthine stimulation I used the medulla of a giraffe. The connections to the muscle centers of the neck are three: one in the nucleus of the vagus nerve, one in the substantia reticulosa and one in Roller's nucleus. The descending fibers are (1) descending fibers from the nuclei labyrinthici, (2) fibers from the nucleus interstitialis and (3) fibers from the nucleus ruber, the nucleus reticularis and Deiters' nucleus. All the latter fibers originate from large motor nuclei, influenced, on one hand, by the cerebellum and stem ganglion and, on the other, by the vestibular apparatus. The influence of the cerebellum on the labyrinth is one of degree of movement, tonus and power. From the nucleus interstitialis, the tractus interstiospinalis runs medially in the posterior longitudinal bundle for the righting reflex. The nucleus commisuralis also receives fibers from the vestibular nuclei. From the commisural nucleus the tractus commissurospinalis conveys tonic impulses to the postural muscles.

The conclusion is that in the higher animals there is a finely graded apparatus controlling the ocular muscles and all the muscles of the body. These movements are unconscious, but in higher animals there is a direct connection with the cortex through which they get orientation in space. The disorientation is called dizziness. The vestibular system influences the vegetative system also, chiefly through the vagus nerve.

ANATOMY OF THE CRANIAL BLOOD SINUSES, WITH PARTICULAR REFERENCE TO THE LATERAL. DR. BARNES WOODHALL, Durham, N. C.

There are several theories as to why the right lateral sinus carries more blood than the left (60 per cent plus, right predominance; 10 to 15 per cent, left predominance). Several different types of arrangement of venous outflow from the cranial cavity are classified as follows:

1. Common pool types, the classic textbook picture, actually less common than other types. The longitudinal and the straight sinus meet in a more or less common pool, from which the blood continues into the lateral sinuses, which are approximately equal in size.

2. Plexiform type. The longitudinal and straight sinuses may be each divided into two divisions; the lateral sinuses usually are unequal but communicate with each other. There are many variations of this arrangement.

3. Ipsilateral type. The longitudinal sinus runs to one side, usually the right, and the straight sinus to the opposite side. Communication between the lateral sinuses, if present, must be through an additional channel or a foramen. In most cases the lateral sinus receiving the flow from the longitudinal sinus will be the larger.

4. Unilateral type, rare. Both the longitudinal and straight sinuses empty into one lateral sinus, the other being absent or attenuated and carrying blood only from minor vessels.

5. Occipital Type. The occipital sinus is large enough to be of value in carrying blood; there may be a large single or paired occipital sinus, similar to the fetal, with large marginal sinuses, or the longitudinal or the straight sinus or both may empty into a large occipital sinus at the expense of one lateral sinus.

Cases observed in which autopsy was done clinically illustrate these types and their subvariations. In certain cases observed clinically in which recovery

occurred evidence of intracranial pressure, both general and localized, was presented. The Tobey-Ayer test is related to the venous variations, and roentgenography, particularly the anteroposterior view, is valuable to demonstrate the relative size of the lateral sinuses.

DISCUSSION

STACY R. GUILD, PH.D., Baltimore: There are certain common variations of the relation of the dome of the jugular bulb to the middle ear.

(The variations were illustrated by slides.)

The distance between the floor of the tympanic cavity and the top of the jugular bulb varies greatly. There may be dehiscences in the floor, sometimes so large that almost all the mucosa of the hypotympanum rests on the bulb. When the dehiscences are small crypts of mucosa may project through and extend between the bone and bulb, with the lumen continuous with the middle ear and larger at the distal end. Infection from these crypts may originate primary thrombosis of the bulb. The last two slides show, respectively, attachment of the inferior margin of the tympanic mucous membrane to the jugular bulb and the defect in the ampulla of the posterior semicircular canal. Neither of these conditions produced attributable clinical symptoms.

IMMUNOLOGIC ASPECTS OF BLOOD INVASIONS WITH SPECIAL REFERENCE TO SINUS THROMBOSIS. DR. GREGORY SHWARTZMAN, New York.

Virulence represents a function of two related variables, one the power to invade the host and the other the power of the host to respond to the invasion. Each of these variables depends on a long series of its own variables.

One is given a bacillus recovered from a patient's urine. A mouse's resistance to the infection does not tell one what the patient's resistance is.

Dr. Goldman and I analyzed 168 consecutive blood cultures for Streptococcus haemolyticus.

Two group studies were made:

1. The invaders belonged to the same serologic group, but the portal of entry varied.

2. The portal of entry was the same, but the type of organism was different.

There was a definite relation between the portal of entry and the outcome of the disease.

In presulfanilamide days the mortality rate from pulmonary infections, surgical infections, infections associated with nonbacterial diseases, gynecologic infections and osseous and articular infections with an identical strain varied from 100 per cent from pulmonary infections to 20 per cent from secondary erysipelas complicating an infection, with which the expectation of mortality was not increased.

There was a different strain, infecting mostly children, in early spring or late fall, the infection beginning as a disease of the upper part of the respiratory tract without formation of pus, with local recovery, but uniformly producing osteomyelitis of a long bone. There were few organisms in the blood culture, which cleared rapidly. Operative treatment was a great advantage.

The technic of blood culture is as follows: Twenty-five cubic centimeters of blood is taken, 19 cc. seeded into fluid mediums and 4 to 6 cc. seeded into solid mediums.

The number of original colonies per 5 cc. of blood may be taken as an index of the number of bacteria in the blood stream. (Five cubic centimeters is seeded into each medium.)

According to my experience any organism found in the blood stream after surgical mastoiditis is interpreted as indicating involvement of the lateral sinus.

If solid mediums alone were used, the positive cultures would drop 50 per cent; if only one fluid medium instead of three were used, they would drop two thirds.

This applies only to Str. haemolyticus. With Streptococcus viridans one may observe a transitory and innocuous invasion.

Why does the streptococcus obtain dominance in the blood stream over other invaders of the middle ear? How does it penetrate the three lines of defense—mucosa, mastoid bone and vessels?

The pathogenic group, classified as A by Mansfield on a basis of specific carbohydrates (B, C, D, E, F, and G are nonpathogenic for man) produces an enzyme-like substance, which is fibrolytic for human fibrin. Some types also can enhance the permeability of human tissues to toxins. The toxic factors precede the actual invaders, cause changes in the venous wall and produce a sterile thrombus preceding invasion by the bacteria themselves. The bacteria which can dissolve the thrombus and prevent organization will be able to invade the blood stream. Any secondary thrombus formation will then be associated with more severe phlebitis. I cannot conceive how ligation is of benefit. My idea is to limit the area of infection by promoting organization and allowing the fibrin to acquire immunity.

Infection by the hematogenous route produces severe damage. The effects of local infections or reinfections are slighter.

THE PREVENTION OF DEAFNESS IN CHILDREN. DR. SAMUEL J. CROWE, Baltimore.

When a child with no evident impairment of hearing for speech and no subjective aural symptoms is found to have retracted tympanic membranes and an abnormal amount of lymphoid tissue around the pharyngeal orifice of the eustachian tubes, the audiometric test will invariably show loss of hearing for tones at the upper end of the scale. At first the mucosa of the middle ear is congested and the loss is only for tones far above the speech range. Later the mucosa becomes myxomatous and the child has difficulty in hearing speech. The retraction, at first limited to the membrana flaccida, extends to the anterior quadrant and then to the entire tympanic membrane. This statement is based on a series of audiometric tests on children of from 7 to 12 years. Lymphoid tissue is more abundant before puberty. Removal of tonsils and adenoids does not remove lymphoid nodules in the wall of the eustachian tube and the pharynx and, in fact, may result in their secondary formation. They are best treated by radiation. The diagnosis can be made only by using the nasopharyngoscope. If the examination cannot be made with the area under local anesthesia, avertin with amylene hydrate should be used. Loss of hearing for high tones alone does not always indicate a lesion of the inner ear, nor does loss of perception by bone conduction for a 512 double vibration fork. Retraction of Shrapnell's membrane is a danger signal. Tonsils and adenoids are removed far too frequently in children. In order to safeguard the hearing it is necessary to make examinations of the drum membrane, the pharyngeal end of the eustachian tube and the acuity of hearing for the higher frequencies once or twice a year till puberty. In addition to irradiation I advise using sulfanilamide during acute infection.

DISCUSSION

DR. BURT R. SHURLY, Detroit: In Detroit a test of school children showed deficient hearing in 4.8 per cent. In clinics many operations for adenoids are done by novices; final inspection is not done, and poor results are overlooked. The La Force type of adenotome removes only the tissue in the midline. A resolution was recently introduced at a meeting of the American Medical Association asking the help of members in making a nationwide setup for the assistance of children with defective hearing, and the American Otological Society should encourage that. In Detroit there is a follow-up system, and neglected deficiencies are referred back to the family doctor.

In the hurried clinic the examination may be superficial and many things undiscovered. I believe that the key is early diagnosis in the school system and that problems of nutrition are important. I hope that research workers will explain why the lymphoid tissue develops as it does.

DR. EUGENE R. LEWIS, Los Angeles: The ideal is to prevent rather than cure, but prevention has aspects of intangibility. It takes an unusual spirit to

cast his lot where the more he accomplishes the less it shows. What has been prevented is not susceptible of demonstration.

No matter how much transitory impairment is caused by disease, the possibility of restoration of function must be kept to the fore. Recoverability must be supreme among clinical considerations, and conservation of normal equipment and function must be the goal.

Dr. William Mithoefer, Cincinnati: There is no small area in the body as rich in veins and nerves as the lateral wall of the pharynx and the fossa of Rosenmüller. The veins of the eustachian tube communicate with the nasopharynx and the middle ear. Pressure in the nasopharynx causes congestion of the middle ear. Congestion of the membrana tympani often disappears after adenoidectomy or after application of cocaine and epinephrine. It is a circulatory disturbance, and therefore hearing for high tones was depreciated in Dr. Crowe's tests.

This is not only a local but a constitutional disturbance, and one should think of hypothyroidism as a frequent disease of childhood and also of hypoglycemia, vitamin deficiency and allergy.

Dr. Rafael Lorente de Nó, New York: Can the treeatment be done by an otologist, or must it be done by a radiologist?

Dr. E. E. Carmody, Denver: How about the use of radium salt?

Dr. Horace Newhart, Minneapolis: Will Dr. Crowe please explain the limitations of the ordinary phonograph audiometer in disclosing deficiencies of hearing for higher frequencies?

Dr. Samuel J. Crowe, Baltimore: The nasopharynx is irradiated with an applicator small enough to pass with ease along the floor of the nose. A radon-containing glass tube is enclosed in a brass tip, which acts as a screen. One end is closed; the inside bore is 1.5 mm.; the total length is about 1.5 cm.; the outside diameter is 2 mm., and the thickness of the wall is 0.5 mm. The applicator will hold 50 mg. of radium sulfate or 1,000 millicuries of radon. A safe dose for lymphoid tissue in the nasopharynx of a child is 1.5 to 1.8 gram minutes on each side for the first treatment; a month later 2 to 2.5 gram minutes on each side may be given if necessary. The dose in gram minutes is calculated by multiplying the number of millicuries in the applicator by the number of minutes the applicator is in the nasopharynx.[1] Thus, if the applicator contains 600 millicuries, the minimum exposure is two and one-half minutes and the maximum four and one-fourth minutes on each side.

One must test with an audiometer giving a pure tone up to 16000 double vibrations. When the speech range is involved the deafness is already advanced. No method of treatment avails if the child is 16 or 18 years of age. The solution of the problem in school children is to detect the loss of hearing long before it can be detected with the phonographic audiometer.

Head Noises and Deafness: Peripheral and Central. Dr. Edmund P. Fowler, New York.

I shall present a method for differentiating peripheral from central tinnitus and deafness and also a method for detecting and measuring certain effects of subaudible and audible tinnitus.

Sustained tinnitus is variously described by different patients. Measurements of its loudness vary from a few decibels above threshold to 45 decibels or more. Tinnitus should be measured for its loudness, its masking by applied frequencies and its masking effect on applied frequencies. There are two kinds of tinnitus, vibratory and nonvibratory. Vibratory tinnitus is caused by actual vibrations reaching the end organs in the cochlea; it is always peripheral; it may occur in normal ears; it is masked by extraneous sounds because it is set up by actual

1. Ed. Note.—This gives the dose in millicurie minutes; 1 grain minute equals 1,000 milligram minutes, which is equivalent to 1,000 millicurie minutes.

vibrations reaching the cochlea; when present it may lower the hearing by both air and bone as a masking noise does; the effect on bone conduction is constant and important. Vibratory tinnitus is made up predominantly of lower frequencies.

Nonvibratory tinnitus is caused by other factors, such as biochemical stimulation, and is wholly due to factors operating on the neural mechanism. They may be vasomotor. Chemical changes or variations in pressure along the course of the auditory nerve may change its firing rate. Clinically such changes may be evidenced by the Ménière symptom complex. It is independent of vibrations in the cochlea. It may be peripheral or central. Usually higher-pitched sounds predominate. Common excitements are disturbances of the gastrointestinal tract, of the ear itself, of the nose, of the throat or of the teeth. Smoking and temporomandibular dysfunction are causes. The latter is caused by activation of the nerve elements. (A slide was shown.)

Tinnitus must be studied in relation to the following factors: The excitability level of the nervous system and psyche; the type of the underlying factors; the presence and amount of "recruitment of loudness"; if it is vibratory, the predominance of air or of bone conduction, and the summation and masking effect from and on it of a different frequency band coincidentally present. Central tinnitus is masked with difficulty because the projected pattern differs from a peripheral stimulation. Moreover, masking sounds do not get through; they are stopped by the central lesion. Severe obstructive deafness will prevent extraneous sounds from masking tinnitus by air conduction.

These studies have an important bearing on the etiology, prevention and treatment of the disorders causing deafness.

DISCUSSION

Dr. Rafael Lorente de Nó, New York: In 1931 Professor Alien made the observation that the cut end of a nerve is the source of a continuous stream of impulses similar to physiologic stimulation. This traumatic discharge explains such symptoms as pain. A few years later Dr. Gasser found that the discharge could be controlled by chemical means (calcium) and also by changes of potential, which may be recorded. (A slide was presented showing that a change of p_H changes the number of nerve impulses.) If ionization of calcium is present, the discharge is increased enormously by adding citrate solution. The nerve is now cut down to one fiber. (A slide was presented.) A rhythmic series of impulses at a high rate, continuing for hours is shown. The citrate is washed off, and the nerve ceases firing except once for each stimulus, which is normal. If the calcium is removed, the nerve ceases firing, but the ganglion is fired spontaneously. If the calcium is restored, the initial state is restored. If there is a disturbance of the acoustic nerve, the impulse produced by the cochlea is followed by another force, which will be heard as a noise. If the disturbance increases, the nerve will produce impulses in succession, and that will be tinnitus. If the tones are due to the nerve acting spontaneously, they can be modified by impulses from the periphery. If the tinnitus is due, e. g., to chemical irritation it will not be affected by a tone delivered to the ear. The laboratory experiment is a nice model of tinnitus.

R. L. Wegel, Ph.D., New York: It appears that for the first time otologists have a quantitative objective measurement of tinnitus. Dr. Fowler has refrained from detailed generalization on account of paucity of data. A provisional subdivision of the cases of his second class of tinnitus would be desirable. The suggested groups are, first, cases in which the tinnitus or its cause obliterates the character of the perceived sound and, second, those in which the interference is no more than an equal amount of noise to a normal ear, that is, cases, respectively, in which each of these conditions predominates over the other. In the first group distortion occurs only at the lower intensities, between threshold and 40 decibels above it. Distortion is limited to a narrow band, one octave in the cases so far reported. The range is different in different ears (only 2 cases available). As the intensity of the test tone is increased, the point at which the

noise is replaced by a sensation of a pure tone is much sharper than at the threshold. Cases of acute tinnitus seem to fall most characteristically into this group. The outstanding contribution in Dr. Fowler's paper is the outline of a quantitative objective technic of measurement of tinnitus.

DR. MAX A. GOLDSTEIN, St. Louis: In a case of otosclerosis in which the tone frequency of the tinnitus has been definitely obtained, why after further loss of hearing has occurred is there a complete cessation of tinnitus?

DR. EDMUND P. FOWLER, New York: Tinnitus always covers a band of frequencies, and many things can change its character. Sometimes simply taking the patient into the test room does it. I had tinnitus from the explosion of a gun some years ago; in a half-hour it disappeared, but after a few years it reappeared with variations. I thought probably it was caused by sclerotic arteries or something else irritating the neural mechanism. I went into the sound-proof room, and it disappeared. It came back later in the day, and I have been able to measure it and its masking. This has happened to several patients. It takes little sometimes to change or stop tinnitus. I now wish to show a roentgenographic motion picture of a jaw joint in passive and active motion. The jaw swings on a broad fulcrum. The head has a back and forth sliding movement; it is pushed down only when it rides up onto the tubercle in marked extension. The angle and ramus swing in an arc like the movable jaw of a biting instrument and give the illusion that the condyle is moving vertically. It rotates clockwise and counterclockwise about a vertical axis in grinding motions. On the side with loss of molars and one bicuspid there was local discomfort and loss of hearing in the tonal areas of tinnitus that was present. The patient avoided discomfort by biting on the other side and not clamping down hard on the stem of his pipe.

THE PROGRESS OF DEAFNESS IN CLINICAL OTOSCLEROSIS. C. C. BUNCH, PH.D., St. Louis.

(Sixteen slides were shown illustrating hearing tests of a series of patients with a diagnosis of otosclerosis lasting over six years.)

The diagnoses in this series were made by trained otologists on the basis of findings with tuning forks, supplemented by audiometric tests which I performed. In no case was the diagnosis based on the audiometric test alone. The progress of the disease varies. It may be rapid, slow, intermittent or stationary or even improve over a long period. The best method of obtaining commensurate values is by the audiometer. The results of hearing tests following operation for otosclerosis recorded only for whisper and voice, unless the distances are great, are not reliable. Sleep, nervousness, fatigue and psychic factors, all play a part. If the patient "wills" to hear, the distance becomes greater. If he is tired, the effort becomes too great, and he quits trying to hear.

Some of the graphs show improvement for some frequencies, loss for others and repeated crisscrossing of the results of various tests at different times. In this study changes greater than plus or minus 5 to 7.5 decibels are considered greater than may be explained by the factor of attention, and a 10 decibel error is allowed for the observational factor. A nurse, told to study lip reading, has improved and continues her duties, and her loss of hearing is not noticed by outsiders. Six women who bore children show no loss. Two who underwent major operations show improvement.

Six years is too small a part of a lifetime to enable one to draw conclusions. It seems certain that time alone is not the factor determining the progress of the disease. With those in whom deafness increased no obvious factor in health or in the tests gave clues on which predictions could be based. Changes in either direction were often unilateral. The patients' own statements as to gain or loss were often not substantiated by the tests.

DISCUSSION

DR. EDMUND P. FOWLER, New York: I agree with Dr. Bunch. I have observed many patients over fourteen or fifteen years, with a condition diagnosed as otosclerosis, in whom there has apparently been no more change than in the average person not having this disease. Hearing fluctuates up and down but varies little from year to year. When obstructive deafness affects sounds of 40 or 50 decibels advancing nerve deafness may develop with it. There are many interrelating factors to consider, but one should not harbor a defeatist attitude toward one's patients. In many instances the otologist who foretells progressive and severe deafness is wrong.

THE USE AND EFFECTIVENESS OF HEARING AIDS. DR. GORDON BERRY, Worcester, Mass.

I shall take up the subject from the standpoints of the physicist, the otologists and the patient. (In the two latter capacities Dr. Berry is personally qualified and in the first more so than many otologists. A. L.) There is considerable variation of intensity between different parts of speech and between accented and unaccented syllables. The masking effect of extraneous noises must also be considered. The more severe the deafness the narrower the band between the threshold of hearing and painful noise. Normal speech ranges between 90 and 8000 cycles, but the most important frequencies are between 500 and 2000 cycles. The telephone is limited to 300 to 2500 cycles. Two types of portable hearing aid are now available. The carbon granule type has the advantage of lightness and smaller cost of upkeep; it has been longer in the market and is better standardized. It is usually adequate for a 20 to 60 decibel loss of hearing. But its magnification is limited to the frequencies between 300 and 3000 cycles, with a sudden peak at which noises are too loud when others are not loud enough; also it overemphasizes background noises. The vacuum tube type permits more even magnification for all speech frequencies, with less distortion, is more powerful and permits more selective amplification for different types of deafness. Generally it is more suitable for patients with more than a 50 decibel loss of hearing and for those with inner ear deafness. The bone conduction type may be preferred for conduction deafness; the air conduction, for nerve deafness. The bone conduction instrument requires more amplification but is less likely to cause pain when high intensities are used—for some find it necessary in order to hear the faint consonants to set the rheostat so high that loud sounds are almost painful. The ear piece is usually preferred by men and by those who use a box instrument. It can be slipped in and out more easily, while the spring headband is more easily concealed by women. It is best to have the fitted earpiece, when this is used, of hard material, both for hearing and for comfort. Wet batteries which can be recharged may be used when it is desired to maintain a high voltage but are larger and of greater initial cost. Stationary sets can be made to plug in on street current. A high grade battery concern should make batteries to fit all types of connection and overcome the individual monopoly each company tries to institute by unique connections. More than one company is offering to deal directly with the physician, and another offers considerable reduction if the profit to the middle man can be so saved. According to my own experience there is a process of adjustment in the use of a hearing aid—the sounds at first seem strange; so a conscientious trial is necessary.

Most patients need an aid long before they will accept one. The otologist should not make an issue of it but should have the patient try one "for occasional use." A trial before purchase helps. At this stage it is the speaker's "poor diction" or the "poor acoustics of the hall," not the patient's deafness, that is at fault. A visit to the League for the Hard of Hearing to see members using aids without embarrassment helps. Increasing deafness makes the patient's earning ability precarious. I believe that wearing a hearing aid is not a barrier to business contacts.

On the contrary, mishearing and an erroneous reply create the idea of stupidity or deafness and strain the relation. The ear phone makes him more efficient and honest with his environment. When the deafness is so severe that an electric aid is inadequate the hearing tube should be used, and the patient is fortunate indeed who has learned lip reading early, before this severe stage has been reached.

A hearing aid does no harm. On the contrary the effort to use it keeps the acoustic function keener, but one must avoid amplification to the point of painful sensation. Before the final purchase the hearing with the aid should be checked by the otologist, and it should have been tried out first in the patient's normal environment. After considering the audiometer test, the graduated vocal test under calibrated control and tests under a normal environment, I prefer for practical purposes sentence tests with the spoken voice under a normal environment. The hearing aid should increase the distance at which sounds can be heard about five times at least.

In conclusion I wish to stress the acoustic gain and personal comfort that accrue from a properly fitted hearing aid, and I believe that the otologist is in a strategic position to guide the patient and relieve him of worry. The making of hearing aids is becoming an exact science, and I bespeak the support and encouragement of the members of the American Otological Society.

DISCUSSION

HARVEY FLETCHER, PH.D., New York: I believe the carbon set can be made to do as well as the vacuum tube set. It all depends on the kind of carbon microphone and the kind of vacuum tube set in question. My guess is that the future will see more carbon sets, because they are capable of functional improvement and of being made smaller and more convenient.

Bone conduction receivers are less efficient than air conduction receivers by about 40 decibels, so the patient must have that much better hearing by bone to get the same result with the same power. If the loss of hearing is 80 decibels, there is probably nerve deafness, and it is unlikely that the bone conduction will be 40 decibles greater than that by air. The sentence test is the most practical. Twenty sentences are sufficient.

DR. HORACE NEWHART, Minneapolis: It has become an important function of the otologist to help his aurally handicapped patients. The number of persons who can be helped has been increased. The otologist who recommends a given device without investigation risks discrediting himself and the manufacturer. He should recommend the type best suited to the patient, charge a reasonable fee and never accept a commission or bonus in violation of medical ethics. The patient should be allowed a period of trial on the basis of a reasonable rental. This procedure is not favorably regarded by the average salesman, but he should see the wisdom of permitting this privilege. The otologist should cooperate understandingly with the ethical vendor of "accepted" hearing aids. The patient should understand that the hearing aid is not a complete solution of his problem and that effort should be made to conserve his residual hearing by contact with his physician. Patients should also be warned against exploitation by unscrupulous dealers. The possibility of litigation on account of real or alleged unskilful attempts to obtain a cast for the earpiece should be remembered.

DR. KENNETH M. DAY, Pittsburgh: During the past five years I have experienced an increased loss of hearing for high tones, due, I believe, to overuse of my hearing aid. I have only a narrow margin between the threshold and a sensation of pain. This probably occurs only in cases of fixation of the stapes, as there is no buffer against loud jarring noises. I wear a receiver in each ear and with less amplification get an increased intelligibility of 30 per cent. I can better differentiate conflicting sounds, but with both ears plugged I had to reeducate my voice. I hear better by air conduction because of loss of bone conduc-

tion for the higher tones. Distortion of his own voice, by plugging the ear, leads a patient to believe mistakingly that he hears better by bone, besides being more conspicuous.

Dr. Edmund P. Fowler, New York: Did the hearers notice how clear were the voices of Dr. Berry and Dr. Day? The hearing aid is a great help to the hard of hearing in modulation of the voice.

The Use of the Monochord in Routine Tests of Hearing. Dr. Frederick T. Hill, Waterville, Me.

Bunch says of all the devices available for determining the upper tone limit he considers the monochord the most satisfactory for both air and bone conduction. It has the same amplitude for both air and bone. The tuning fork stem used for bone conduction has only one-hundredth the amplitude of the prongs. The audiometer does not have its intensity for high tones, and those ordinarily used do not extend above 8192 double vibrations. The monochord is normally heard by bone 2000 double vibrations above the level at which it is heard by air conduction. With advancing age there is a loss for high tones of 1000 to 2000 double vibrations per decade above the third, at which time the normal range is 20,000 (approximately); toxemias and otitis media may cause loss for high tones. That this is not merely mechanical is shown by the fact that the loss by bone is in the same ratio. There are no differences after removal of impacted cerumen or paracentesis; consequently I believe that lesions of the middle ear may also cause degenerative processes in the internal ear, which are made manifest by the monochord. Crowe has shown that loss of hearing for high tones is an early symptom of a pathologic condition of the eustachian tube and occurs with loss of ossicular mobility. It may also be a symptom of acoustic tumor or Ménière's disease. Loss of hearing for high tones by bone conduction is more indicative of nerve deafness than loss of hearing by air. The ear not being tested must be carefully masked. With chronic suppurative otitis media one usually finds loss along the entire range by the audiometer. This may be of good prognostic value if the hearing for high tones is preserved by bone conduction.

DISCUSSION

Dr. D. Harold Walker, Boston: Any help in measuring the hearing by bone conduction is valuable in obtaining information regarding the condition of the internal ear. The monochord may prove of great value in determining the advisability of fenestration of the semicircular canal if that operation becomes established.

C. C. Bunch, Ph.D., St. Louis: The tone of the monochord is intense and difficult to control for measurement. This handicap must be overcome if one is to use the monochord effectively. The audiometer, the tone of which is measurable, does not give sufficient intensity in the high frequencies. In the soundproof room the fan may be turned on without any masking of the monochord. When the patient cannot distinguish between the "squeak" and the rubbing one knows one is reaching the high tone limit. With the Galton whistle the hiss is confused with the whistle. With the Koenig rod the thud of the hammer may be difficult for the clinic patient to distinguish from the ring. Two Bárány noise apparatus often fail to mask the squeak of the monochord; nevertheless it is the most valuable of the three instruments.

Dr. Werner Mueller, Boston: The monochord is musically important as a way station between the bowstring and the grand piano. Scientifically it has no peer as a producer of accurately measurable tones. As an audiometric instrument it has not had the place it deserves. It is simple in construction and manipulation. The length of the vibrating portion of the steel wire is set by a sliding metal block, and the pitch read off by the numbers engraved on the side

of the ruler. It lacks the adventitious noises of the Galton whistle or the König rods. It can be used in testing for bone conduction. If kept free from rust it retains its tone almost indefinitely. If the string breaks it is easily replaced. The size of the string and its tension make little difference. The pitch is determined by the speed with which sound will travel in steel. The original monochord had a range of from 6000 double vibrations up. The Schaefer modification, by using either a bow or a hammer for transverse vibrations brings this as low as 435 double vibrations. (It goes even lower. A. L.)

Dr. Frederick T. Hill, Waterville, Me.: The patient I mentioned was deaf for high tones in one ear; so masking was not an important factor.

The Endaural Route. Dr. D. E. Staunton Wishart, Toronto, Canada.

A triangle of skin is removed from the posterior, external and superior portion of the canal, which is not cartilaginous, the aperture enlarged by a vertical incision in the incisura auris anterior and the periosteum pushed back, and then, by using two hand retractors and pulling on one and relaxing the other according to the part desired uncovered, the entire field can be exposed. Unusual illumination is necessary. Difficulty may be encountered in exposing the retrosinal cells and those of the tip if they are far back. Particularly difficult is this approach if the soft tissues are swollen or scarred. Hemorrhage is another handicap. The assistant who holds the retractors must be skilful and have endurance. The immediate dressing is simple, only pockets, if any, requiring packing for drainage. The after-care makes great demands on the operating surgeon, as the meatal region is sensitive and the dressings cannot be properly done by a nurse or intern. When excessive granulations occur, the healing is especially tedious. The period of hospitalization averaged considerably more than in cases of postauricular incision. The comments of patients, 2 of whom had also had operation by the postaural route, were generally favorable to the intra-aural approach. The vertical incision, if continued into the temporalis muscle, may introduce infection. It must be packed open. (In 1 case in which it was sutured abscess developed.) Occasionally scarring occurs, and, if so, it is conspicuous as compared with a scar hidden by the auricle. The operation should not be undertaken by a surgeon not specially trained for it and even in expert hands requires more time. This approach gives better visualization of the middle ear and is therefore to be preferred for the fenestration operation. However, I prefer the postaural incision for simple mastoidectomy, which does not require visualization of the middle ear, and for most radical mastoidectomies, which require removal of much diseased tissue. The skin flap of the endaural method can be so manipulated as to make a satisfactory graft for the facial ridge and mastoid bowl in the radical operation.

Whiting's rules for the modern mastoidectomy call for "complete eradication of all diseased bone, in the shortest time commensurate with thoroughness, by methods designed to reduce to a minimum dangers incident to the operation and calculated as well to induce speedy and permanent healing." I conclude that the endaural route is not the method of choice for simple or radical mastoidectomy.

DISCUSSION

Dr. James A. Babbitt, Philadelphia: I am in agreement with the statement of the necessity for great care in incision, the advisability of postauricular incision in operation for acute mastoiditis, the requirement of the personal attention of the operating surgeon to postoperative dressings and the advisability of having any secondary mastoiditis which may arise operated for by one who understands the endaural route. I have not observed that there is danger of introducing infection into the temporal fascia, that the procedure takes longer than operation by the postauricular route or that it has produced secondary perichondritis. If the periosteum is well lowered there is good exposure of the tip and the postsinal regions. Granulations may be due to careless injury to the posterior wall. They are disturbing and

long lasting, and their treatment, rather than the difficulties in procedure, should be emphasized. Besides excluding this approach for simple mastoidectomy, I should favor its use in radical operation in cases in which precise work is required in the middle ear and in fistulization.

DR. D. E. STAUNTON WISHART, Toronto, Canada: I had only 1 case of infection in the temporal region; still, with all respect to Dr. Babbitt, I think I was fortunate. His criticism that I have not sufficiently emphasized the treatment of granulation is correct. It is of great practical importance.

RADICAL MASTOIDECTOMY; USE OF THE TENSOR TYMPANI MUSCLE IN CLOSING THE EUSTACHIAN TUBE. HENRY M. GOODYEAR, Cincinnati.

In every simple mastoidectomy drainage from the antrum through the posterior wound should be maintained until the middle ear is healed. Cholesteatoma with a small perforation in the attic in children should be treated with a modified radical operation. Even with a dry ear in such a case intracranial complications may suddenly develop. When the attic is involved I remove the external wall of the attic with a Richards curet until the epitympanic recess is fully exposed and communicates with the antrum. The malleus is visible but is not amputated. The outer matrix of the cholesteatoma and the ear drum are disturbed as little as possible. For a high posterior perforation the dissection is carried forward only to include the perforation. The incus is removed if necessary. A circular skin flap according to the technic of Whiting is used and the cavity packed with iodoform. For the complete radical operation I prefer a burr and a round curet to avoid possible petechial hemorrhages into the labyrinth. The table is tilted 25 degrees for convenience, and the anterior and inferior walls of the canal are curetted to widen the tympanic cavity and give better access to the mouth of the eustachian tube. The tensor tympani muscle is identified by its ligament; the processus cochleariformis is broken down and the muscle tucked into the mouth of the tube as a transplant. The muscle is usually more than large enough. I have used this method for eighteen years. The methods of others may be cited, but I believe that mine succeeds best, as the muscle is left attached to its blood supply as a live graft.

DISCUSSION

DR. LEROY A. SCHALL, Boston: Dr. Goodyear's statement that the appearance of the tympanum and not the appearance of the posterior wound should be the guide to the progress of the healing should be drilled into every house officer. There is no mention of the diagnosis of cholesteatoma by roentgen rays. By the usual plates cholesteatoma can be diagnosed in 45 per cent of cases; with the base plate, in an additional 25 or 30 per cent. In 25 per cent of cases it cannot be demonstrated, as there is as yet no erosion of bone. I believe the endaural route will become more popular with better selection of cases. The use of the tensor tympani muscle to close the tube is logical and should be practical. It was formerly thought that many failures to obtain a dry ear were due to a patent tube, but many patients with a dry ear have an open tube. The better selection of cases for tympanal mastoidectomy has been responsible for this. The recurring infection of the tympanic cavity due to a pathologic condition in the nasopharynx is not treated by this type of operation.

SULFANILAMIDE AND ROENTGEN RAY THERAPY FOR ACUTE OTITIS MEDIA AND MASTOIDITIS. DR. FLETCHER D. WOODWARD, Charlottesville, Va.

A statistical survey is made for eight years before the use of sulfanilamide and for two years thereafter. The dose was ½ grain (0.03 Gm.) per pound (0.5 Kg.) of body weight per twenty-four hours. Occasionally the drug was used parenterally. There have been some untoward reactions but no serious complications. Sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was used in few instances. My associates and I feel that surgical indications should not be

influenced by the administration of this drug. There have been some dramatic cures of streptococcic and pneumococcic infections, and there should be no hesitation in rendering to Domagk full credit for his original work. Our good results were with certain types of Streptococcus and Pneumococcus, which fortunately are responsible for the majority of aural infections. Staphylococcus, Streptococcus viridans, Bacillus proteus, Bacillus hofmanni, Bacillus influenzae, Bacillus tuberculosis and diphtheroids were not influenced. The ratio of mastoidectomy to otitis media was reduced, as were complications except meningitis, of which there were 8 instances with 6 deaths. In all these the condition either was present on admission, was due to organisms not influenced by sulfanilamide, occurred in patients in poor general condition or was of an unusually fulminating type. One recovery was from infection with Pneumococcus type III and 1 from petrositis, epidural abscess and sinus thrombosis due to Streptococcus haemolyticus.

In 34 cases acute otitis media was treated roentgenologically. We did not feel that such treatment was of any value for this condition. It was of benefit for reduction of lymphoid tissue in the nasopharynx and about the orifice of the eustachian tube, for the control of granulation tissue after radical mastoidectomy and for control of polyps and granulation tissue in chronic purulent otitis media.

DISCUSSION

Dr. John R. Page, New York: It is difficult as yet to evaluate the results of the use of sulfanilamide from the records of the Manhattan Eye and Ear Hospital. In 1934, without sulfanilamide, 938 ears were submitted to myringotomy and 369 to mastoidectomy; i. e., 39 per cent required operation. In 1937, with the use of sulfanilamide, there were 1,029 ears submitted to myringotomy and 389 to simple mastoidectomy, or 38 per cent operated on. These results are not far apart. The mastoidectomy record is so high for both years that I think the attacks must have been unusually severe, for in my own service among 836 myringotomies in another year without sulfanilamide there were 43 mastoidectomies with 1 death, 5.1 per cent. This is as good a record as that of Horan and French, cited by Dr. Woodward. For meningitis cures have been effected that would not have been obtained without it. Sulfanilamide has confused otologists' interpretation of mild involvement of the mastoid, as it has roentgenologists'. Apparently the drug is not as efficacious in the bone as elsewhere, and one must be careful not to be deceived. It happens more frequently now that the tympanum apparently clears up and the infection in the mastoid continues to smolder. The drug is tricky, and I do not use it for ambulatory patients. I wish to report on a patient who had mastoidectomy in childhood but experienced continued discharge. He was treated by roentgen rays thirty years after, but he had daily rise of temperature, headache, loss of weight, odorless discharge and no evidence of cholesteatoma. On reoperation a completely reformed cortex was encountered; the antrum was dry and lined with atrophic membrane; the attic was open and free; the cancellous portion was bloodless; one softened area extended deep into the petrous bone. In the lower part of the mastoid several large cells were filled with pus, and the contiguous sinal wall looked infected. Apparently absorption was from there. Prompt recovery occurred. The events in this case seem to show that roentgen rays had an effect on granulation and on the mucoperiosteum of the mastoid cells but did not stop the infection in the lower part of the mastoid. They probably promote drainage by this selective action on granulations and swollen mucosa, but are contraindicated for advanced involvement of the mastoid. Until the roentgen therapeutists learn more about clinical otology and the otologists more about roentgen therapy, I shall continue to take my chance with a good clinical otologist.

Studies of the Waltzing Guinea Pig. Dr. Moses H. Lurie, Boston.

The waltzing guinea pig tends to run in circles either to the right or to the left. It has a fine tremor of the head and is deaf. These characteristics are inherited and follow the mendelian law for a recessive characteristic. The righting

reflexes are normal. The eyes do not respond to vestibular stimulation. The eyeball is normal histologically. The vestibular portion of the labyrinth is histologically normal. The saccule does not degenerate with the cochlea. No evidence was obtained that the saccule had anything to do with the reception of sound. These observations indicate that the lesion is a central nervous one, and it is believed to be in the region of the red nucleus. The principal abnormal histologic observation is progressive degeneration of the organ of Corti, beginning at the first and second turns and progressing toward the helicotrema. Studies of intrauterine and newborn animals showed that the lesions were not due to congenital lack of development but were degenerative. The changes were less marked in the cochlear nerve and the spiral ganglion. Experimental cutting has shown that the cochlear nerve can degenerate without degeneration of the organ of Corti. The animal develops a normal end organ which later degenerates. This is attributed to a hereditary factor, and perhaps human deafness can also be due to a hereditary degenerative factor not necessarily otosclerosis.

EXPERIMENTALLY INDUCED CIRCLING (WALTZING) IN THE GUINEA PIG. DR. MOSES H. LURIE, Boston, and DR. E. W. DEMPSEY, Boston.

Cutting the eighth nerve or destruction of one labyrinth in the guinea pig did not produce the characteristic phenomena of waltzing.

The animals did not show nystagmus on circling or movement of the head.

Enucleation of the eyeballs of the waltzing guinea pigs did not stop the waltzing. The eyes were histologically normal.

Righting reflexes were normal.

The vestibular labyrinth was histologically normal.

The vestibular nerve and its medullar endings were normal. In experimental operations it was found that to produce circling the lesion must be placed just caudal to the mamillary bodies. The operative mortality was exceedingly high, hemorrhage being the chief cause. All the animals that recover after anesthesia move in circles. If the lesion is in the left side of the brain, circling is to the right (clockwise). The animals can go in a straight line. After some days there is beginning compensation. Two animals that survived now show dilatation of the left pupil, fixation of the left eyeball, a tendency to turn to the right and tilting of the head to the right. The third survivor showed only the circling. We believe that the cause of the circling is a break in the reflex between the vestibular apparatus, the nucleus of the third nerve and the postural centers. A lesion in the region of the interpeduncular nuclei may cause symptoms similar to those seen in the waltzing guinea pig.

DISCUSSION

STACY R. GUILD, PH.D., Baltimore: I object to the term "waltzing." The behavior is similar to that of other animals and birds which can walk straight and eat normally at times and at other times undergo forced movements. I do not question that the lesion in the experiments is in the interpeduncular nucleus, but it involves other structures. Can it be that the neck reflexes are the cause? One can fasten a pigeon's head back and get peculiar forced movements. I agree that there is cochlear degeneration rather than malformation, but there is concomitant degeneration in the cells of the external sulcus in areas in which the end organ is atrophied. In man this seems to precede degeneration in the organ of Corti. Dr. Crowe, Dr. Polvogt and I described this in the "Pathology of High Tone Deafness." I do not know the relation. I doubt if this investigation gives any clues as to Ménière's symptom complex. I do not think Dr. Lurie meant to imply that every one believes that the only form of hereditary deafness is otosclerosis. There is a rich literature describing other forms of hereditary deafness.

DR. WILLIAM J. MCNALLY, Montreal, Canada: Dr. Lurie mentioned shaker guinea pigs. It is possible to produce shaker frogs by removing the semicircular canals and leaving the utricles intact. The animals are greatly disturbed, and their

heads keep bobbing up and down uncontrollably. One of the difficulties in interpreting labyrinthine reactions is that the distribution within the brain stem of the nerves from the different end organs is not known. It is possible that Dr. Lurie's animals have a lesion of the brain stem, possibly involving canals with intact utricles, and it may be that if he and Dr. Dempsey can localize that lesion they will be among the first to tell something about the distribution in the brain stem of the nerves from the end organs of the canals, on the one hand, and the vestibule, on the other.

Dr. Moses H. Lurie, Boston: I am sorry Dr. Lorente de Nó could not be present. "Waltzing" is the term used in genetics to describe this animal. It acts like the so-called waltzing mice. The question of neck reflexes is interesting, because Professor Ibsen has written me that he has found a shaker guinea pig. The head of a shaker mouse goes up and down when he is excited, and there is a tendency to fall backward. Dr. Dempsey has removed structures anterior to the lesion; he has removed the whole of the mamillary bodies, and it is only when the lesion is caudal to the mamillary bodies and in the interpeduncular nucleus that the circling syndrome occurs. In animals which survived removal of half the midbrain there was no circling. I think it is important to get rid of the misconception held by some that any progressive deafness with a hereditary history is otosclerosis, especially in children, who may be totally deaf in one ear when one cannot find a reason for it. I wish to thank Dr. Guild for raising important points on interpretation of facts, for, after all, he did not read the paper. One of the great difficulties in working with animals with inherited tendencies is that one observes no gross lesions. In the brains of waltzing guinea pigs, using different types of nerve stains, Dr. Dempsey and I were unable to distinguish the regions of the vestibular nuclei from those of normal animals.

Skull Fractures Involving the Ear. Dr. W. E. Grove, Milwaukee.

Two hundred and eleven cases of fracture of the skull among 1,187 cases of injury to the head were studied in detail after hospitalization. Also a group of 152 cases of injury to the head including 17 cases of fracture of the skull were studied within twenty-four hours of admission to the hospital. Of the latter group damage to the ear appeared in 49.

In the group of 211 there were 146 cases of longitudinal and 16 of transverse fracture of the temporal bone and 49 of fracture of the vault or miscellaneous fractures. Briefly, longitudinal fracture is a lesion of the middle fossa through the tegmina antri and tympani and the middle ear and sometimes through the canal. It is frequently associated with bleeding from the ear, and if the dura is torn, with leakage of cerebrospinal fluid. Bleeding has been known to occur late, even four weeks after injury. Transverse fracture is a lesion of the posterior fossa, crossing the pyramid and involving the labyrinthine capsule and its contents and often the facial nerve. There is loss of function and spontaneous nystagmus. It may be combined with longitudinal fracture, and then bleeding from the ear occurs. Fractures may be microscopic and not demonstrable except by histologic methods. It is possible to have a transverse fracture without total destruction of function. It is possible also to have injury to the head with destruction of the function of the inner ear without fracture. Because much of the labyrinthine capsule is formed of enchondral bone, healing largely takes place by fibrous union instead of bony, and there is always danger of late meningitis, even years later, if otitis media occurs in a case in which there is also a communication with the middle ear. In addition to the symptoms mentioned, hemotympanum, hydrotympanum, ecchymosis in the area of the mastoid several days after injury and radiologic evidence of fracture may be found. For visualization of transverse fracture the Stenvers position is best, and the roentgenogram need not be taken early. For longitudinal fracture the picture should be made as soon after injury as possible. Even with several exposures many such fractures will be missed.

Paralysis of the facial nerve occurred with 31 per cent of transverse and 18 per cent of longitudinal fractures. It was permanent with 1 transverse and 5 longitudinal fractures. In the remainder it improved or cleared up entirely. Immediate paralysis is due to a lesion of the nerve. Late paralysis is due to hemorrhage into the canal and secondary neuritis. Cure is the rule but may take several months.

Cochlear function cannot be determined immediately because of cloudiness of the sensorium. Later, as many such cases are medicolegal, one must be on guard for malingering and bear in mind that one does not have knowledge of the previous state of the hearing. With longitudinal fracture the damage was greater in general in older people. Some showed no loss of hearing, but many showed a perceptive type of deafness and in many cases equal loss in both ears. This indicates that commotio labyrinthi exists, if one understands by this hemorrhage into the inner ear. In all my 16 cases of transverse fracture there was total loss of hearing on the affected side. On the contralateral side 3 patients had normal hearing, and 3 had slight and 7 moderate to subtotal loss. Fractures which do not directly involve the temporal bone may affect the hearing, and this occurs almost in direct relation to the age of the patient. With longitudinal fracture there is usually a combined type instead of a pure conduction type of deafness. In approximately 75 per cent of all cases of fracture of the skull vertigo was complained of, and the irritation was diagnosed as central in origin.

The mortality from basal fractures of the skull averages 55 per cent. Eighty-five per cent of the deaths occur in the first forty-eight hours. About 15 per cent occur later, usually of meningitis, and this may occur after apparent convalescence; death even sixteen years after a transverse fracture is reported. Nine of my patients who discharged cerebrospinal fluid recovered, which controverts the black outlook given this symptom by Davis. As to management of the condition in the ear, I recommend noninterference, the ear being protected with sterile cotton or gauze. If suppuration is present or supervenes with longitudinal fracture the usual surgical indications for mastoidectomy are awaited. With transverse fracture suppuration calls for immediate intervention with exposure of the dura and with drainage of the labyrinth if there is any evidence of meningitis.

DISCUSSION

Dr. Alfred Lewy, Chicago: The subject of Dr. Grove's monograph is important from the standpoint both of the immediate management of the condition and of the medicolegal aspect of the sequelae. The subject is inadequately handled in the literature. In particular, discussion of the precise indications for and technic of surgical intervention is lacking or is controversial. The frequency of otologic complications with fracture of the skull shows the importance of otologic consultation in cases of severe injury to the head.

I suggest a clinical classification of such injuries, as follows:

1. Injuries to the head involving the middle and the external ear.

2. Injuries to the head involving the internal ear and the vestibular system, (*a*) with escape of cerebrospinal fluid, (*b*) without escape of cerebrospinal fluid, (*c*) with otitis media present at time of injury and (*d*) with otitis media developing after injury.

Patients with injuries involving only the middle or the external ear are operated on only for uncontrollable hemorrhage (which is rare) or for otitis media on the usual indications for mastoidectomy. Those with injuries involving the internal ear are operated on immediately after shock is overcome if purulent otitis media is present or on its advent. If there has been escape of cerebrospinal fluid the tear in the dura should be uncovered and a Neumann labyrinthectomy performed. If meningitis is present the internal auditory meatus should be included, the cisterna pontis lateralis thus being drained. If no escape of cerebrospinal fluid and no open communication exists with the internal ear a Hinsberg operation

may be added to the radical mastoidectomy. New developments in chemotherapy may modify the present recommendations. If definite evidence of communicating fracture is lacking a more conservative course may be justified.

If routine exposures plus a basal plate do not disclose a suspected fracture, the Stenvers position for transverse and the Mayer position for longitudinal fractures are recommended.

A Survey of the Use of Sulfanilamide for Acute Otitis Media. Dr. Karl M. Houser, Philadelphia.

Questionnaires were sent to each member of the American Otological Society, to two hundred and fifty-five other otologists in the United States and Canada and to one hundred and fifty pediatricians. One hundred and eighty-four answers were returned, representing thirty-seven states and Canada. Of those replying 89.6 per cent use sulfanilamide for otitis media; of these 60 per cent believe it should be used early, 58.5 per cent not even awaiting identification of the infecting organism. The total number of cases reported was 9,667. The mortality was 0.041 per cent. Seventy-six per cent of those replying believe that the incidence of acute mastoiditis was reduced. Only 46 have had experience with control cases and none with a sufficient number to permit drawing reliable conclusions. Twenty-seven per cent used the drug for the nasopharyngitis preceding otitis media. Corr and Root stated that the smallest amount of sulfanilamide reported to have been administered before the onset of granulocytopenia was 25 Gm. and concluded that a total distributed dose of 20 Gm. is safe. The average administration was ¾ to 1 grain (0.05 to 0.06 Gm.) per pound (0.5 Kg.) of body weight the first twenty-four hours, decreased as the condition permitted. Frequent examinations of the blood should be made. The fact must be recognized that sulfanilamide will not cure an infected mastoid that has undergone necrotic change. According to Lockwood, the efficiency of this drug depends on the ability of the blood stream to distribute blood to the infected parts, which it cannot do to necrotic areas. In the University of Pennsylvania Hospital the incidence of surgical mastoiditis has not changed in three years, but there the patients when admitted are usually suffering from advanced conditions.

Ninety per cent of the physicians reporting are using sulfanilamide. Perhaps mostly the enthusiasts replied.

Sixty per cent believe that it reduces the incidence of acute mastoiditis, and the majority believe that it should be used early.

If a pneumococcus is found or its presence suspected, the preference should be sulfapyridine.

The concentration in the blood is probably not important.

The survey shows that the use of sulfanilamide for otitis media is widespread and that the users are enthusiastic. This enthusiasm lacks the support of control cases.

DISCUSSION

Dr. Harry P. Schenck, Philadelphia: The value of the drug against most beta hemolytic infections has been established, but its use for otitis media remains controversial. Dr. Houser's figures indicate which way the wind is blowing. The survey brings out the lack of control and of bacteriologic studies. If it were only possible to determine the organisms in the cases in which the drug appears ineffective, statistics would acquire more significance. For pneumococcic infections both sulfanilamide and serum should be used, because the drug is only bacteriostatic, while the serum neutralizes the toxins. Satisfactory results cannot be expected when the influenza bacillus or Staphylococcus is the infectious agent. Sulfanilamide does not eliminate the necessity for paracentesis or surgical drainage, regardless of the location of the abscess. Three peculiarities of the drug deserve reemphasis: Fever due to the drug may make differential diagnosis difficult; withdrawal may be followed by exacerbation not controlled

by readministration of the drug; an atypical clinical course may result in bizarre surgical and roentgenologic findings. The effect of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) against pneumococcic and other infections deserves further study.

Dr. Henry M. Goodyear, Cincinnati: Sulfanilamide sometimes causes leukocytosis. In 1 case a leukocyte count of 85,000, with 92 per cent lymphocytes, and in another a count of 80,000, returned to more normal proportions after discontinuance of its administration. If sulfanilamide in sufficient doses does not bring results in three or four days, there is no use in continuing to administer it. It may be dangerous.

Dr. Ralph A. Fenton, Portland, Ore.: It was important to find out what the pediatricians are doing. Under sulfanilamide therapy the symptoms may subside; the child apparently gets well, but three or four weeks later violent complications appear. My associates and I have observed such cases, and it usually turned out that the pediatrician had relied entirely on the drug. When used, the drug should also be administered regularly through the night to keep up the concentration in the blood.

Dr. Alfred Lewy, Chicago: The general statement that sulfanilamide is not useful against pneumococcic infections is incorrect. There are a number of cases of pneumonia due to infection with Pneumococcus type III, and I know of 1 case of meningitis of this type in which the patient recovered under sulfanilamide therapy. It may be necessary to differentiate cases for chemotherapy a little more closely.

Dr. Frederick M. Law, New York: It has been proved that the drug has no effect on absorption of bone. It masks the symptoms, and a single film is insufficient. The absorption occurs in the deeper cells, and with a single film one may visualize only the cells immediately beneath the cortex. One should have a stereographic pair, preferably at different angles. These cannot be projected on the screen; so I advise the otologist to go to the roentgen room and familiarize himself with stereoscopic technic and interpretation.

Dr. Frederick T. Hill, Waterville, Me.: If sulfanilamide is bacteriostatic rather than bactericidal it will lower the virulence of the organism, with consequent lowering of the resistance of the patient. One must keep in mind possible sequelae due to lowered resistance.

Dr. Karl M. Houser, Philadelphia: It was not my intention to leave the impression that sulfanilamide is not efficient against pneumococcic infections, but in a well controlled series of cases my associates and I found sulfapyridine superior against pneumococcic infections, and we give it the preference when we know this is the infective agent.

Report of Unusual Bacterial Findings in a Case of Fatal Chronic Otitis Media with Complications. Dr. Horace Newhart, Minneapolis.

The isolation of a rarely encountered anaerobic, gram-negative, non-spore-forming micro-organism of the genus Bacteroides, from the blood of a patient dying of otogenous sepsis, warrants a report for the record.

The scant literature is reviewed. The common lesions with which bacteroides have been associated have been inflammatory ones of the nose, ear, pharynx, tonsils and urinary and intestinal tracts. The present case is that of a farmer aged 32 with a chronic discharge from the ear since he was 4 years of age. He had had one operation, probably for a polyp. He noticed pain radiating from the ear on July 1, 1938; on July 17 he had a chill accompanied by sweating and vomiting. On July 24 he consulted a physician and was hospitalized. The scleras were icteric, the pupils and the ocular movements normal and the disks perhaps slightly hazy; the hemoglobin content was 79 per cent and the leukocyte count 16,000; diplococci and staphylococci were present in the aural discharge. The tempera-

ture flucuated from 98 to 103 F. Sixty grains (3.9 Gm.) of sulfanilamide was given daily. The icterus became more marked, the neck rigid and the tip of the left mastoid tender. In the left ear a whisper was heard at 2 inches (5 cm.) and in the right at 15 feet (4.6 meters). Perception in the Weber test was lateralized to the left. The left membrana tympani was perforated in the posterior half; foul pus was coming from between pale granulations. The liver and spleen were palpable. No vertigo or disturbance of equilibrium was present. There were pain in the chest and a severe cough producing scant sputum with many organisms, one of which was Bacillus fragilis. The facies was normal. The right mastoid was well pneumatized, and the left had no cellular development but an area of lessened density posterior to the antrum; the chest was clear except for accentuated bronchial markings. Spinal puncture revealed yellowish fluid with 5 cells per cubic millimeter and no pus cells; culture revealed gram-positive micrococci, which were regarded as a contamination. Subsequent cultures of blood revealed colonies of B. fragilis. The patient improved temporarily but was worse on August 5, and an exploratory operation revealed extensive destruction of bone, wide exposure of the dura, which was bile stained, an organized perisinal abscess and no lumen. Many bacteria were found, but anaerobic cultures showed B. fragilis. The patient experienced marked euphoria. He died on August 21.

Autopsy revealed a cerebellar abscess on the right (contralateral), purulent basal meningitis, old thrombi of the superior sagittal and petrosal sinuses, bronchopneumonia, three small abscesses (essentials only mentioned) and three small hepatic abscesses.

It is my belief that infection with bacteroides would be revealed in more cases if there was more general resort to anaerobic cultures.

DISCUSSION

DR. JAMES G. DWYER, New York: The moral is that in cases of chronic discharge from the ears one should make anaerobic studies on material from the ears and on the blood. The organism is not a primary invader and does not attack a previously healthy person. The same is true of Bacillus pyocyaneus. If anaerobic cultures were made in cases of abscess of the brain and of sinus thrombosis higher bacteria would probably be found. The presence of these bacteria is analogous to that of Streptococcus in actinomycosis of the lung. The icterus, due to metastases to the liver, is interesting.

THE FREQUENCY OF INCREASED NYSTAGMUS TIME AFTER ROTATION IN CASES OF MULTIPLE SCLEROSIS. DR. PAGE NORTHINGTON, New York.

The report represents a survey of 2,000 case reports. To induce nystagmus by rotation ten turns were used in twenty seconds. In the cases of spontaneous nystagmus the postrotatory nystagmus was observed with the eyes in the position in which the spontaneous nystagmus was not apparent. The literature is reviewed. Duration of nystagmus for more than thirty seconds is considered hyperactivity. Records are cited of over 200 student aviators with only 3 having nystagmus beyond this limit; Malan reported among 11,000 normal subjects only 7.12 per cent in whom nystagmus lasted over thirty seconds. The average for the army is twenty-four seconds, with thirty-four seconds as the upper limit. The results showed that prolonged postrotatory nystagmus occurred with multiple sclerosis in 77 per cent of the cases, with psychoneurosis in 19 per cent, with arteriosclerosis in 22 per cent and with intracranial tumors in 27 per cent.

Spontaneous nystagmus occurred with multiple sclerosis in 63 per cent, with psychoneurosis in 18 per cent, with arteriosclerosis in 18 per cent and with intracranial tumors in 42 per cent.

The hearing of patients with multiple sclerosis compared favorably with the hearing of a normal group.

Prolonged postrotatory nystagmus occurs with multiple sclerosis with greater frequency even than spontaneous nystagmus.

Further observation is being made in cases of prolonged postrotatory nystagmus in which the condition was diagnosed as psychoneurosis. Some of the patients may prove to have an organic lesion.

DISCUSSION

DR. STANLEY M. DILLENBERG, New York: Multiple sclerosis is being used as a scrap basket for numerous neurologic syndromes. Charcot's syndrome is seldom seen early. Symptoms referable to the pyramidal tract are often missing, and abdominal reflexes are lost in only 80 per cent of cases. Prolonged postrotatory nystagmus, present in 77 per cent, compares favorably with the absence of abdominal reflexes as a diagnostic sign. Urinalysis and examinations of spinal fluid, including the colloidal gold and other chemical tests, usually leave one in doubt. At present research is in progress to find a means of determining abnormalities in the cholesterol, lecithin and esterase content of the blood and spinal fluid or a serologic test similar to the Wassermann. A carefully taken history helps. The course is usually intermittent, with temporary dysesthesia, diplopia, blindness, numbness and fleeting disturbances of the sphincter. If the future confirms Dr. Northington's observation, a diagnostic weapon of no little importance is provided. The pathologic picture of multiple sclerosis is degenerative demyelinization; hence I agree that the prolonged nystagmus is due to removal of inhibition and is central. The psychoneurotic group with prolonged nystagmus is being followed. Only 2 patients have been checked so far, and they do not show evidence of an organic lesion.

REPORT OF THE CHICAGO COMMITTEE ON OTOGENIC MENINGITIS, 1939. DR. ALFRED LEWY, Chicago.

The report this year was intended to deal with pneumococcic meningitis. Because of interest, two other conditions are included:

1. In the case of a boy 1 year of age, the type IV pneumococcus was cultured from the spinal fluid and blood. Bilateral mastoidectomy was performed. Type IV rabbit serum and sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) were given. The patient recovered.

2. Meningitis due to the type XVIII pneumococcus followed a sphenoethmoid operation. Type XVIII rabbit serum was given intraspinally and intravenously, with sulfapyridine therapy, intraspinal injections of air and blood transfusions. The patient recovered.

3. A girl aged 10 years suffered from a septic condition with elevation of temperature. The ears were at first normal, but a Tobey-Ayer test indicated block of the left jugular vein and roentgen examination showed coalescent mastoiditis on the left. Mastoidectomy was performed with removal of a purulent thrombus in the lateral sinus and ligation of the jugular vein. Culture of the spinal fluid showed Streptococcus viridans. There was intermittent fever, the temperature sometimes reaching normal, and once the spinal fluid was sterile. Later apicectomy was performed. Therapy was attempted at first with sulfanilamide and later with sulfapyridine; blood transfusions were given. The course of the disease was fluctuating and prolonged, with a downward tendency. Cerebellar exploration gave negative results. The patient died after several months. No postmortem examination was performed.

4. A 20 year old woman had mastoiditis and meningitis; mastoidectomy was performed. Culture of material from the mastoid showed Streptococcus haemolyticus and that from the spinal fluid a gram-positive diplococcus, believed to be a mutation form of the streptococcus. Sulfanilamide therapy resulted in recovery.

5. A 3 year old boy had mastoiditis and meningitis. Culture from spinal fluid showed type III pneumococcus. Mastoidectomy and sulfanilamide therapy brought about recovery.

6. Four patients with pnemococcic meningitis not otogenous recovered under sulfapyridine therapy.

Conclusions.—Sulfanilamide appears to be useful against the type III pneumococcus. For infection of the meninges with other pneumococci sulfapyridine appears to be the remedy of choice, supplemented by the appropriate serum, which should in part be administered intraspinally. Perhaps intravenous use of sodium sulfapyridine will obviate gastric distress. Intraspinal injections of air may have value in preventing spinal block.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM AMICITIÆ SACRUM

President: Dr. Louis Ledoux, Brussels, Belgium.

Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

HUNGARIAN OTOLARYNGOLOGICAL SOCIETY

President: Dr. V. Zimányi, Zárda-u. 48, Budapest II.

Secretary: Dr. G. Kelemen, Reáltanoda-u., Budapest IV.

SOCIEDAD RIOPLATENSE DE OTO-RHINO-LARINGOLOGÍA (ARGENTINE SECTION)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.

Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

SOCIÉTÉ FRANÇAISE D'OTO-RHINO-LARYNGOLOGIE

Secretary: Dr. Henri Flurin, 19 Avenue Mac-Mahon, Paris, 17e.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. A. W. Proetz, 3720 Washington Blvd., St. Louis.

Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.

Place: New York. Time: June 10-14, 1940.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.

Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. Lyman Richards, 319 Longwood Ave., Boston.

Secretary: Dr. Paul H. Holinger, 1150 N. State St., Chicago.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

Place: Westchester Country Club, Rye, N. Y. Time: May 27-29, 1940.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lee M. Hurd, 39 E. 50th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Waldorf-Astoria Hotel, New York. Time: June 6-8, 1940.

SECTIONS:

Eastern.—Chairman: Dr. John R. Simpson, Medical Arts Bldg., Pittsburgh.
Place: Pittsburgh. Time: Jan. 5, 1940.
Southern.—Chairman: Dr. Walter J. Bristow, Doctors Bldg., Columbia, S. C.
Place: Columbia, S. C. Time: Jan. 8-9, 1940.
Middle.—Chairman: Dr. Sam E. Roberts, Professional Bldg., Kansas City, Mo.
Place: Kansas City, Mo. Time: Jan. 19, 1940.
Western.—Chairman: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles. Time: Jan. 26-27, 1940.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Horace Newhart, 527 Medical Arts Bldg., Minneapolis.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Westchester Country Club, Rye, N. Y. Time: May 30-31, 1940.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY EXAMINATION

The American Board of Otolaryngology will hold an examination at the Manhattan Eye, Ear and Throat Hospital, New York, June 3, 4 and 5, 1940.